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Nutrition in the Newborn*

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INTRODUCTION

During the past decade there has been a tremendous reduction in feeding difficulties and in nutritional disorders. The improved nutritional status along with other factors has had a share in the progressive lowering of mortality rates in infectious and other diseases.

A quarter of a century ago the pediatrician was defined frequently as the "baby feeder"; a variety of milk mixtures, compounded in many instances of skimmed milk, whey, and cream according to percentage and adjusted to meet supposed indications in the baby, constituted his chief stock in trade. His activities in preventive pediatrics were linked with "milk stations," "diet kitchens," and "babies' milk fund foundations."

The pediatrician's other medical duties in the field of nutrition chiefly called him to battle with summer complaint, an engagement in which he was all too frequently defeated. His weapons were tea, barley water, protein milk, and floating and seaside hospitals.

How changed is all this today! More facts, discovered in biochemical laboratories and applied by scientifically trained physicians, have established infant feeding on a really sound basis.

Vomiting is now a less frequently observed symptom, and while poor nutrition in infants is still observed, the terms "balance disturbance," "infantile atrophy," and "infantile dystrophy" are disappearing from pediatric literature in many parts of the country, although the conditions may still be prevalent in foreign countries where recent advances in the nutrition of the newborn have not been studied. Today we do not split hairs over slight changes in amounts of protein, carbohydrate, and fat, nor do we argue much about the relative values of casein and whey proteins.

CHEMICAL DIFFERENCES BETWEEN COW'S MILK AND HUMAN MILK

It is necessary, however, in order to understand the nutritional processes of babies fed on cow's milk, to have a clear idea of the essential ways in which cow's milk differs chemically from human milk.

Fats.—I shall briefly review the role of fats in infant nutrition. The fats of the two milks differ considerably in their chemical nature. The fat of human milk contains only about 2 per cent of the lower irritating volatile fatty acids, that of cow's milk contains anywhere from 6 to 27 per cent, according to different observers. It is possible that this high content in irritating acids, especially butyric and caproic, may have a great deal to do with the difficulty some infants have in digesting cow's milk.

Of the more volatile acids, oleic makes up at least 50 per cent of the total fatty acid content of human milk fat, whereas it makes up only 35 per cent of the total content of cow's milk fat. Cow's milk fat contains much more palmitin than does that of human milk. The calcium soaps of oleic acid are much more easily absorbed than those of palmitic acid. This may be another reason the fat of human milk is tolerated better than that of cow's milk. The fat globules of human milk are probably in a much finer emulsion than those in cow's milk. When the number of droplets are counted in a counting chamber there are always more in human than in cow's milk. The fat globules in human milk measure between 0.001 and 0.01 mm. Since the fat droplets in human milk may be of greater diameter than those in cow's milk, it seems inconsistent that there should be a larger number in the former than in the latter. The explanation must be that the majority of fat droplets in human milk are small and measure about 0.001 mm., while the majority of those in cow's milk must be closer to the upper limit and measure nearly 0.01 mm.[1] The larger the number of fat globules, the greater is the surface area for the fat-splitting ferments to work on, and, therefore, the more complete is the digestion of the fat. This may be another reason the fat of human milk is better as an infant food than that of cow's milk.

Protein and Salts.—One additional consideration of the diet in infancy is the subject of caloric proportions. From the analysis of the chemical composition of human breast milk it is logical to conclude that an ideal diet for the infant should provide the essential fatty acids such as linoleic. The remaining common long chain fatty acids, both saturated and unsaturated, are relatively nonspecific in their nutritive behavior, and the evidence is strong at the moment to support the belief that there are no specific fatty acids other than the essential fatty acids required in human nutrition.

It is known that cow's milk contains more than twice as much protein and more than three times as much ash as human milk. This is because of the more rapid growth of the calf, which needs a large amount of protein and salts in order to supply material for the rapid growth of bone and muscle tissue.

It is interesting to note that in most Mammalia the ash and protein content of the milk of each species is in inverse proportion to the length of time needed for the young to double its birth weight.[2]

Most of the protein in cow's milk is casein, in the proportion of about 6 parts of casein to 1 of lactalbumin. In human milk, according to most authors, the casein and lactalbumin are about equally divided.

The casein of cow's milk is probably not the same substance chemically as the casein of human milk. Casein in cow's milk contains 0.87 per cent of phosphorus; casein of human milk contains only 0.25 per cent. Cow's milk casein coagulates in the stomach in tough, leathery masses, human milk casein in fine flakes.

The salt content of cow's milk is greatly in excess of that of human milk. Although as yet relatively little is known regarding the metabolism of salts and their role in producing disturbances of digestion or nutrition, it is undoubtedly true that the large amount of inorganic material, particularly salts of calcium, sodium, and phosphorus, and the forms in which they are found, have a great deal to do with some of the disturbances in babies fed on cow's milk. Cow's milk is, however, much poorer in iron than human milk, and

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this, too, has an important bearing on practical infant feeding, because babies fed too long on milk, without other iron-containing food, are likely to become anemic and flabby.

It is important to note that the high salt content in cow's milk in relation to fat digestion favors the formation of insoluble calcium soaps which are difficult to absorb.

Summary.—

1. There are important physical and chemical differences between the fats in cow's milk and human milk which indicate that the former is digested and absorbed with more difficulty than the latter.

2. The total protein is considerably higher in cow's milk, owing to the large amount of casein present. It is a different substance chemically from that in human milk and is probably digested with more difficulty.

3. The salt content of cow's milk is very high in comparison with that of human milk. The large excess of calcium salts seems to be of special importance since it probably has much to do with disturbances of fat metabolism.

INFANT FEEDING AND DIGESTIVE AND NUTRITIONAL DISTURBANCES

With these differences in mind it is not hard to see why the metabolic processes of breast-fed and artificially fed babies are very different, and why it is possible for a totally different category of digestive and nutritional disturbances to develop in artificially fed and in breast-fed infants.

To many pediatricians, discussions of the relative merits of lactose, cane sugar, corn syrup, Dextri-maltose, and oatmeal gruel are outmoded, and malt soup and butter-flour mixtures are assigned to oblivion.

The lowering of digestive function and tolerance for food during an infection is a condition more clearly recognized now than it was a few decades ago. We know that the most frequent causes of vomiting in the past were unmodified milk protein and too much fluid bulk; rarely were the causes too much fat, sugar, or casein. In large measure because of the outstanding work of Americans, the importance of protein modification is definitely recognized. While protein modification can be accomplished in a variety of ways, no one of them is better or easier than simple cooking, a procedure accomplished by boiling, drying, or evaporating, often followed by canning.

It is recognized that the caloric intake is variable from feeding to feeding, but the total can be estimated fairly accurately, and, in the long run, the intake is pretty largely determined by the child's appetite. It is also recognized that 15 to 20 per cent of the total caloric intake should be in protein, 40 to 50 per cent in carbohydrate, and the remainder in fat. Furthermore, if water is added to a milk mixture it should be done to meet the child's water requirement and not for the sole purpose of making the food more easily digestible.

In the early years of this quarter of a century, human milk was a sine qua non of even reasonable success in infant feeding, but the nutritional importance of human milk is no longer dominant in infant feeding. It is true, as it always has been, that human milk is easily assimilated, cheap, clean, and convenient, and for these reasons breast feeding should be energetically fostered. However, it is also true that science has met in large measure the challenge of producing a nutritionally acceptable substitute for human milk. The dried milk powders on the market today are very useful and can be used to prepare a balanced diet for the newborn.

Pediatrics has made great strides in eliminating summer complaint and noninfectious diarrhea in babies. The morbidity from these complaints is now negligible. Many factors of course have been operative in bringing this situation to pass, notably improved infant feeding, better nutrition, good nursing, and intelligent medical care guided by the results of brilliant clinical and laboratory studies. These have demonstrated that the symptoms are largely due to fluid loss, electrolyte imbalance, and vascular and circulatory failure. Today therapy consists essentially in discouraging further fluid loss by putting the gastrointestinal tract at rest (feeding nothing by mouth), by fluid and electrolyte replacement, and by antishock treatment; no procedure, in this latter respect, is as effective as blood transfusion. When the body and the intestinal tract are restored to a near normal condition the patient can be fed (at first in small quantities), and the food need not differ from that which would be given under ordinary circumstances. If oral intake of food must be held in abeyance for several days organic substances other than glucose, namely, vitamins and amino acids, can be administered parenterally.

VITAMINS

The vitamin chapter in the science of nutrition and in pediatric practice is likewise remarkable. Vitamins play an important role in infant nutrition. Many of the most important problems of prevention of rickets, tetany, xerophthalmia, and scurvy have been solved. As recently as 15 to 20 years ago, the majority of the cases admitted to children's wards were either diarrhea or the results of vitamin D deficiency, rickets, and convulsions due to rachitic tetany. Now all these disorders are preventable.

Recently Gardner[3] pointed out that the calcium-phosphorus ratio in milk should be 1½ to 1. By application of this knowledge to infant-feeding problems, early convulsions in the newborn have almost entirely disappeared. There is danger of precipitating this condition by feeding an evaporated milk formula. Whole milk, with a calcium-phosphorus ratio of 1 to 1, is in itself on the danger threshold. When the formula is further diluted to 1 oz. of evaporated or cow's milk to 2 oz. of water or even 2 parts of milk to 1 part of water, the food is unphysiologic for the human infant in the neonatal period. This dilution produces elevated serum phosphorus and decreased serum calcium and magnesium levels. In addition, a high phosphorus diet of a formula of inadequately diluted cow's milk causes hypertrophy of the parathyroid gland of the newborn. If breast milk is unavailable, a suitable formula of simulated breast milk is advisable for the neonatal period.

With the early addition of vitamins to the formula and newer biochemical evidence regarding the required milk composition, we do not see, as formerly, dozens of babies with frank convulsions and dozens of newborns having "fits" the moment they have a degree of fever or cut a tooth. The cases of active and latent tetany, such as spasmophilia, are now a rarity, and with the disappearance of tetany has vanished, among other things, the fine art of determining electrical reactions.

There is no necessity for a prolonged discussion of the vitamins and their role in pediatric practice, with the exception of the role of B_{12} in relation to growth and development and B_6, which only recently has come under discussion.

Vitamin B_{12}.—

In 1948 research workers[4] reported the isolation of vitamin B_{12}. Following this discovery APF (animal protein factor) concentrates were produced by fermentation processes as by-products in the manufacture of streptomycin and other antibiotics.

The APF concentrates were tested as supplements to poultry rations, and it was observed that these APF concentrates had feeding value equal to vitamin B_{12} and to protein from animal sources.

The successful experiments in growth promotion in animals prompted Wetzel[5] in 1949 to study the association of growth failure in school children with vitamin B_{12} deficiency. It must be pointed out, however, that he studied these under constraints which parallel those in anemia, in the sense that his results were appraised, not under normal blood or growth conditions, but in hemopathologic states and in conditions of growth failure. The few deficiency cases he reported showed good results; there were no normal cases for comparison. In our own studies[6] on growth and development in the newborn, 346 infants to whom vitamin B_{12} was administered over a 2-year period showed a slight increase in growth as compared to normal newborns.

In appraising the present status of vitamin B_{12}, according to Reisner,[7] 5 to 10 micrograms will give a good reticulocyte response, particularly in pernicious anemia, and larger doses, 25 to 50 micrograms, may have beneficial effects in neurologic lesions. He is also of the opinion that folic acid may be part of or is in part of the B_{12} fraction.

Vitamin B_6.—

An outbreak of convulsive seizures in infants during 1952 and 1953 provided evidence of an essential dietary requirement in infancy for vitamin B_6. Naturally, this aroused renewed interest in the role of vitamin B_6 in human nutrition.

The currently recognized members of the vitamin B_6 complex are pyridoxine, pyridoxal, pyridoxamine, and the phosphorylated forms, pyridoxal phosphate and pyridoxamine phosphate. The functional forms of vitamin B_6 are the phosphorylated compounds, and these esters serve as coenzymes in all the systems involving vitamin B_6.

An important aspect of the metabolic role of vitamin B_6 is concerned with fat metabolism; the synthesis of highly unsaturated fatty acids and the deposition of fat seem to be dependent on an adequate supply of vitamin B_6. Nothing is yet known of the mechanism by which vitamin B_6 brings about these results.

Vitamin B_6 is widely distributed in foodstuffs, the highest levels occurring in yeast and unrefined grains. Processed cereals are low in vitamin B_6, indicating refining losses.

Experimentally it has been demonstrated that a vitamin B_6-deficient diet produces in the animal microcytic anemia and dry skin, followed by dermatitis and, according to recent reports,[8-10] convulsive seizures in infants.

It has been found that the type of carbohydrate in the diet exerts a considerable influence on the composition of the intestinal microflora. Dietary lactose is more favorable to intestinal synthesis of vitamin B_6 than glucose, sucrose, or simple sugars. When sucrose is the carbohydrate, a severe vitamin B_6 deficiency state can be produced in rats. Lactose, dextrose, and cornstarch, however, prevent the development of this deficiency.

In an emergency, 10 to 20 mg. of pyridoxine may be injected intramuscularly or intravenously, and immediate results will be obtained.

The minimum requirement for B_6 is about 100 micrograms per liter; breast milk contains about 110 micrograms of vitamin B_6 per liter.

At a recent pediatric research conference May[11] stated that it would be unfair to pass harsh judgment upon the efforts which were made to improve the one infant food which was associated with the widespread occurrence of convulsions attributed to vitamin B_6 deficiency.

CONCLUSIONS

The thrilling story of the vitamins is again being vitalized by newer developments in respect to chemical isolation and to quantitative determinations of those substances in the human body. Much of the vitamin story has been written in the past quarter-century.

I want to conclude my discussion on nutrition of the newborn by mention of the research concerning liver and iron in the treatment of certain anemias. With modern diet the nutritional anemias so numerous in clinics a few short years ago are disappearing. Every infant should have a blood count at 3 months of age, repeated at intervals of 6 months. Lowering of the hemoglobin during the first few months is a usual finding. Additional iron therapy is recommended, if indicated. However, the reconstructed simulated powdered breast milk on the market today contains sufficient iron to supply the early needs of the newborn before solid iron-containing foods are added to the diet.

As the vitalizing medical discipline for research concerning nutrition of the newborn, pediatrics has certainly justified its existence. And, since all the nutritional problems of infancy are by no means solved, pediatrics in medical schools and hospitals will continue their functions of investigation. However, from each difficulty encountered in an effort to secure ideal nutrition in the infant, we should expect to emerge a little wiser for the experience.

60 Plaza St.

REFERENCES

1. Morse, J. L., and Talbot, F. B.: Diseases of nutrition and infant feeding. Macmillan Co., New York, 1915.
2. Hawk, P. B., Oser, B. L., and Summerson, W. H.: Practical physiological chemistry. Ed. 12. Blakiston Co., Philadelphia, 1947.
3. Gardner, L. I., *et al.*: Etiologic factors in tetany of newly born infants. Pediatrics 5:228-239, Feb. 1950.
4. Jansen, B. C. P.: Isolation of vitamin B_{12} antipernicious anemia factor. Nederl. tijdscher. v. geneesk. 92:2241-2244, July 24, 1948.
5. Wetzel, N. C., *et al.*: Growth failure in school children as associated with vitamin B_{12} deficiency—response to oral therapy. Science 110: 651-653, Dec. 16, 1949.
6. Litchfield, H. R.: Exhibit at International Pediatric Congress, Havana, Cuba, Oct. 1953.
7. Reisner, E. H., Jr.: Present status of vitamin B_{12} in pernicious anemia. Bull. New York Acad. Med. 25:429-433, July 1949.
8. Report of Council on Pharmacy and Chemistry: Pyridoxine hydrochloride (vitamin B_6); status report. J. Am. M. A. 147:322-325, Sept. 22, 1951.
9. Snyderman, S. E., *et al.*: Pyridoxine deficiency in human infant. J. Clin. Nutrition 1:200-207, March-April 1953.
10. Coursin, D. B.: Convulsive seizures in infants with pyridoxine deficient diet. J. Am. M. A. 154:406-408, Jan. 30, 1954.
11. May, C. D.: Round table discussion on vitamin B_6, Chicago, Nov. 1953.

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Craniosynostosis*

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Interest in craniosynostosis, or premature closure of cranial sutures, on the part of the staffs of the Departments of Pediatrics and Radiology at the Grandview Hospital in Dayton, began in April, 1953, when we had the opportunity to observe and study a 5½-month-old female child with this condition. Since that time three additional cases have been observed, and successful surgical procedures have been carried out on two of these children. Prior to this time, in spite of the large accumulation of literature stressing early diagnosis and treatment, we had accepted the problem as one of only limited clinical significance. With the added factor of scarcity of available clinical cases, interest was no more than casual as often happens in dealing with rare syndromes or little understood pathologic problems. Now, in contrast to our casual interest in the comparatively recent past, we are making a constant effort to detect new cases and educate the general staff to the possibility of the anomaly and of its recognition and treatment.

If the disorder is to be handled as one of practical importance rather than regarded as merely another interesting syndrome, it is mandatory that diagnosis and subsequent surgical therapy be carried out at an early age, preferably during the first year and a half of life. It is during this period that the most promising results can be anticipated both for the future skull contour and the mentality of the affected child.

History records examples of unsightly cranial deformities in various races. Hippocrates recognized them as constitutional anomalies. Oxycephaly is pictured in certain Oriental gods representing longevity. In the past century its various forms have been described in detail by many Pan-American and European clinicians. The disease tends to occur in siblings in successive generations and about 85 per cent of cases are reported in male infants.[1]

Craniosynostosis is frequently associated with other defects such as syndactylism of the hands or feet. In such cases it is not infrequent to find solitary involvement of the coronal suture in its entire length. Premature closure of the sutures may be accompanied by other congenital anomalies such as cleft lip and palate, congenital heart disease, spina bifida with meningocele, and abnormalities of the genitalia. There apparently is no racial influence although scaphocephaly appears slightly more common among Negroes. It has not been shown that oxycephaly is more common, as might be expected, in brachycephalic races.[2]

Much difference of opinion regarding etiology is still evident in present-day literature. Virchow (1851)[3] considered it a sequela of meningitis. Other disease processes including syphilis, intrauterine infections, birth trauma, and endocrinopathies have at various times been incriminated as etiologic factors. Some favor the theory of displaced centers of ossification advanced by Rieping[4] who described an oxycephalic skull with ossification centers common to both parietal and frontal bones located in the fused coronal sutures. Hereditary factors have been thought to control the abnormal deposition of such centers. Park and Powers[5] favor the theory of a defect in the interstitial mesenchyme and point out the occurrence of other probable germ plasm defects which so commonly accompany the prematurely stenosed sutures. They feel that the origin of these anomalies is existent in the blastemal skeleton, making craniosynostosis a secondary phenomenon. Congenital sphenoidal defects and hypoplastic changes at the base of the skull as etiologic agents have been generally discounted. Since very few osseus studies are recorded, the etiology is still in question, and the theory of mesenchymal alteration seems the most likely hypothesis to be accepted at this time.

The facial features in craniosynostosis are many times grotesque, and mental retardation occurs in various degrees. This is not difficult to understand when one remembers that the brain increases about 135 per cent in weight during the first year[1] of life and has usually completed 80 per cent of its entire growth by 3 years of age. Also, during the first year more than 50 per cent of all postnatal increases in the circumference of the skull takes place.[6] If normal growth with expansion of the developing central nervous system is directly proportional to the increase in circumference of the growing skull, then brain maturation is possible only in the presence of an ununited, freely expansible vault. In spite of the abnormal or grotesque facial configuration that may occur in an unrecognized case, the thing that is of prime importance is the effect of this bony anomaly on the growth and function of the nervous system. Particularly is this true when obliteration of one or all of the sutures occurs before or shortly after birth. With successful surgical procedures now being carried out for the early correction of craniostenosis, it is highly desirable that all physicians dealing with children be alert to its symptoms and its early recognition.

The fundamental difference between microcephaly and craniosynostosis must be clearly appreciated. In the former it is a failure of the intracranial contents to expand (hypoplasia of the brain) that leads to early obliteration of both sutures and fontanels. The reverse is true in stenosis where occasionally the intracranial hypertension is so marked as to cause noticeable bulging of the anterior fontanel. Differential diagnosis in infancy, especially with the aid of the roentgenologist, is important because of the vast difference in treatment and prognosis.

CLINICAL FEATURES

In the past, much confusion has arisen among physicians regarding the multiplicity of descriptive terms assigned to the various skull abnormalities resulting from premature closure of the sutures. For ease of clinical description only the terms "oxycephaly" and "scaphocephaly" need be employed in most instances. Among some groups, especially roentgenologists, even these terms have been generally discarded in favor of designation of the affected suture and the degree to which it is involved.

The oxycephalic skull, which is turret- or tower-shaped, is due to either partial or complete stenosis of the coronal suture. The former is usually associated with a premature closure of the lateral thirds only and

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therefore does not usually effect an obliteration of the anterior fontanel. A noticeable shortening of the anteroposterior diameter of the skull is almost always evident while the face appears broad and the nose somewhat flattened. Characteristic widening is present in the sphenoid bone and tends to produce a shallow or saucerlike orbital cavity. This factor is responsible for the exophthalmos so frequently seen in the more advanced cases. Radiographic studies of the skull usually reveal an abnormally deep middle and posterior fossa with minimal development of the paranasal sinuses.

Premature fusion of the sagittal suture results in scaphocephaly, which is sometimes referred to as a keeled or boat-shaped skull. Prominence of the narrow forehead and occiput with occasional absence of the parietal eminences is characteristically observed.

A third and less common form is that which occurs when the premature fusion is unilateral, involving the coronal and possibly the squamosal suture. The resulting deformity is a marked flattening of the forehead on the involved side (plagiocephaly).

Because the skull itself is involved initially, the infant or child is frequently referred to the pediatrician because of cranial asymmetry or a vague ocular complaint. The degree of towering, broadening, or elongation of the malformed skull is entirely dependent upon the age at which premature fusion occurred and which suture is involved. If the coronal suture is closed during intrauterine life, the diagnosis may be suspected at birth. However, at this time the deformity is often considered due to severe molding of the head during birth. As Ingraham[1] points out, certainly when abnormal shape of the head does not disappear during the early postnatal period x-ray examination of the skull should be performed without delay to rule out craniostenosis.

Many ocular deformities, including papilledema of the chronic type and subsequent optic atrophy, may be noted in neglected cases in older children. Mechanical displacement of the eyeballs (laterally) with associated squint is not a rare finding in the oxycephalic type of deformity. Complete blindness and dislocation of the eyeballs have been known to occur.[1]

Convulsive disorders due to prolonged increased intracranial pressure are not uncommon in children with craniosynostosis. The seizures may be focal or generalized in nature. The absence of convulsions, or the lack of any signs of central nervous system involvement, for that matter, should not lead the clinician to withhold surgical intervention once the diagnosis is established. It is only by surgical therapy that future mental retardation can be minimized or avoided. It has been repeatedly observed that a dramatic acceleration in development and learning is exhibited by children following craniectomy.

Anteroposterior and lateral roentgenograms of the skull will readily demonstrate which suture or sutures are prematurely fused. Fontanel obliteration is also readily detected, however this is not an absolute necessity in establishing the diagnosis as we have experienced in one of our cases (Case 1). X-ray examination may also reveal marked changes in any one or all of the facial bones. Orbital widening, when associated with early closure of the coronal suture, exophthalmos, and mental retardation, represent a special form of oxycephaly known as craniofacial dysostosis of Crouzon. Oxycephaly may be secondary to other more marked changes in the body such as those seen in the Laurence-Moon-Biedl syndrome in which obesity, retinitis pigmentosa, hypogenitalism, and syndactylism may predominate. A rare form of hemolytic jaundice has also been reported in conjunction with craniosynostosis.[7]

The following two case histories are representative of the most common types of craniostenosis. Successful surgical procedures were carried out on both children. A neglected case of scaphocephaly in a 12-year-old male was also studied; however, surgery was not performed because of the child's age and accompanying mental retardation. It was felt that surgical therapy would not improve either the cosmetic or mental status of this child. At present we have under observation a 14-week-old male infant with bilateral syndactylism and possible premature closing of the coronal suture bilaterally.

CASE HISTORIES

Case 1. A 5½-month-old white female infant was referred by the family physician on April 6, 1953, because of a persistently large anterior fontanel and a tendency toward progressive bilateral exophthalmos. A peculiar facies was first noticed by the parents when the infant was approximately 6 weeks of age. The infant's growth and development, however, had been considered normal in other respects except of late when the parents had noticed increased irritability and excessive crying. She noticed objects, exhibited satisfactory head control, and would sit for short periods with support. There was no history of convulsions, persistent jaundice, or cyanosis. Acute febrile episodes and trauma were denied by the parents.

Past History: The child had been studied at the age of 12 weeks by a physician who recommended air-replacement studies (pneumoencephalography). This procedure was performed and revealed a normal ventricular pattern with no evidence of block. Further studies were not made. The infant was born by ceserean section and the neonatal period was without incident.

Family History: The mother, father, and one other sibling were alive and well. The operative delivery had been performed because of cephalopelvic disproportion.

Physical Examination: Examination revealed a well-developed, well-nourished infant who exhibited an early oxycephalic type of skull. Temperature was 98.6 F., pulse 100, and respiration 22. The forehead was high and broad and a moderate bilateral exophthalmus was present. The anteroposterior diameter of the skull was shortened, and a marked flatness was observed in the region of the occiput. There was a suggestion of hypertelorism. The nose was broad and flat. The anterior fontanel was 8 cm. in width by 7 cm. in length, and the circumference of the head was approximately 2.5 cm. below the normal for her age group (43 cm.). Chest circumference was 44.5 and stem length was 44 cm. Other body measurements were within normal limits. Weight was 16 lbs. and length was 63.5 cm. (50th percentile). Syndactylism or other gross anomalies were not noted. The optic fundi were negative, and results of a neurologic survey were within physiologic limits. Inspection of the nose, throat, and ears failed to reveal any evidence of pathology. The heart and lungs were normal, and no masses were encountered in the abdomen. The findings of the rest of the

physical examination by systems were within normal limits.

Laboratory Examinations: Results of studies, including complete blood count, serum calcium and phosphorus, and urinalysis, were within normal limits.

The infant was referred to the Department of Radiology at Grandview Hospital for a cranial study, particularly evaluation of the sutures. This study revealed questionable premature closure of the coronal suture bilaterally and a slight increase in density in the region of the posterior sagittal suture. There was no evidence of intracerebral calcification or other defects. When the infant was re-evaluated 1 week later the anterior fontanel was under moderate pressure with obvious bulging of the overlying skin. Physical findings were again essentially within normal limits, and the infant appeared to be in no distress. All reflex patterns were still physiologic, and percussion of the skull failed to elicit the "crackpot" phenomenon. The parents thought that protrusion of the eyes had increased slightly since the initial examination. Skull x-rays were repeated and revealed definite evidence of further progressive closure of the coronal sutures. A diagnosis of craniosynostosis involving the coronal sutures was made, and the family was advised of the need for immediate surgical intervention.

The infant was admitted to Cincinnati Children's Hospital on May 26, 1953. The impression of craniosynostosis was confirmed, and on May 29, 1953, a right coronal craniectomy was performed, utilizing polyethylene film. The infant tolerated the procedure well, and a similar surgical correction was made on the opposite side 10 days later. The postoperative course was uncomplicated, and the infant was dismissed 10 days following the second operative procedure.

Serial x-ray studies of the skull have shown no tendency toward bridging of the newly created bone margins. There has been an increase of 5 cm. in the circumference of the skull during the first 13-month period following surgery. The exophthalmos is no longer in evidence, and the cosmetic results are gratifying. There is marked improvement in the facial contours, with less broadening and height of the forehead and visible rounding of the previously flattened occipital area. Periods of irritability have diminished markedly, and growth and mental development have been normal since the craniectomy. The child is walking very well, is beginning to talk, and has no apparent residual impairment of vision. There are no demonstrable neurologic signs or sequelae. The sagittal suture in its posterior aspect is being watched with much interest for possible latent premature closure. The anterior fontanel is closing gradually, although it is still somewhat prominent.

Case 2. A 2½-year-old white male child was referred by the family physician on November 23, 1953, because of the presence of an elevated bonelike mass overlying the region of the anterior fontanel, in association with a long narrow skull. The unusual skull contour was noticed by the parents shortly after birth but they did not become alarmed until a slowly enlarging mass was detected on the vertex. No headaches or convulsive seizures were observed by the family.

Past History: The child exhibited some hesitation in speech, and night terrors had occurred on several occasions. Pregnancy was uncomplicated, and the infant was delivered by brow presentation. The neonatal period was without incident, and the infant was dismissed from the hospital on the seventh postpartum day. One other male sibling, aged 5 years, was living and well. There was no family history of a similar type of skull formation.

Physical Examination: Examination revealed a well-developed, well-nourished male child in no apparent distress. He exhibited a noticeable ridge of bone over the entire course of the sagittal suture and a marked scaphocephalic skull which measured 48.5 cm. in circumference. A circumscribed mass of bone, approximately 5 cm. in diameter, was noted overlying the region of the anterior fontanel. The head was narrowed in its lateral aspect, with a pronounced increase in the anteroposterior diameter. There was no exophthalmos or evidence of facial bone involvement. The posterior fontanel was nonpalpable, and the occiput appeared to be displaced downward and posteriorly. There was no evidence of syndactylism or other congenital defects. Temperature was 98.8 F., pulse 96, respiration 21, and blood pressure 76/54. Chest circumference was 52.5 cm. and stem length 57.5 cm. with the upper-lower ratio being within normal limits. Weight was 16 kg., and length was 37¼ in. Examination of the optic fundi revealed nothing abnormal, and results of neurologic examination were within physiologic limits. Inspection of the nose, throat, and ears did not reveal evidence of pathology. The heart and lungs were normal, and the liver and spleen were not enlarged. Findings of the rest of the physical examination were within normal limits. Blood count, urinalysis, and serum calcium-phosphorus levels were normal.

The child was referred to the Department of Radiology of Grandview Hospital for roentgen evaluation of the skull. This study revealed evidence of premature closure of the entire length of the sagittal suture. There was a slight increase in the convolutional markings of the skull, thought to be caused by increased intracranial pressure. The anterior fossa was relatively short, with increased upward inclination of the floor of the fossa and a peaking of the skull in the region of the bregma. The middle fossa was relatively small while the posterior showed increased elongation as well as increased depth. The coronal, lambdoidal, and temporoparietal sutures appeared patent. There was no evidence of intracerebral calcification. Examination of the cerebrospinal fluid was not performed after the obvious bony defect was revealed.

A diagnosis of craniosynostosis of the sagittal suture was made, and the parents were advised of the desirability for surgical intervention. The child was admitted to Cincinnati Children's Hospital. On January 30, 1954, a sagittal craniectomy was performed, the opposing bone edges being lined with polyethylene film. The patient tolerated the procedure satisfactorily, and the postoperative period was uncomplicated. One month following dismissal from the hospital an increase of 4 cm. was noted in the circumference of the skull. Three months following surgery a total increase of 7.5 cm. in skull circumference was noted. There was almost immediate improvement in the child's speech and cessation of night terrors. Serial x-ray studies of the skull at intervals of 6 weeks have revealed no bridging at the operative site. There are no signs of increased intracranial pressure. A slow increase in lateral expansion of the skull is taking place with cosmetic improvement. The resected mass of bone mounding up

over the bregma has shown no tendency towards recurrence.

DISCUSSION

Since Lane[8] first attempted craniotomy in 1892 for the relief of cranial deformity due to premature closure of the sutures, surgical results have been generally unsatisfactory. Temporary alleviation of the increased intracranial pressure was the most that could be hoped for because of the rapid regrowth of osseous tissue which bridged the newly created artificial sutures. It remained for Ingraham and his associates[9] to demonstrate that strips of polyethylene film placed over the edges of the newly created bone margins would greatly reduce regeneration of new bone and subsequent need for repeated surgery.

Physicians who deal with infants and children should include careful clinical evaluation of the skull as an important part of the total body examination. Normal skull configurations and their abnormal variants must be clearly appreciated in order to recognize the early osseous deformities of craniosynostosis and thus prevent irreversible damage to the nervous system in an otherwise normal infant or child. It must be stressed, however, that recognition during early infancy is most desirable for successful treatment and satisfactory end results, the preservation of normal mentality, and prevention of gross skull abnormalities.

Routine skull x-ray studies are to be recommended in any newborn infant exhibiting syndactylism of either the hands or the feet. Any infant in whom there is sudden or gradual failure of the normal skull expansion, as determined by periodic recordings of skull circumference, may be a candidate for craniosynostosis. This should be strongly considered in the differential diagnosis, especially if an older sibling has been affected. The presence of long hair, particularly in female children, may disguise early clinical manifestations so that diagnosis may be delayed beyond the optimal time for surgical intervention. Conversely, any infant or child with proved manifestations of craniostenosis should be carefully surveyed for the presence of congenital anomalies elsewhere in the body.

SUMMARY

1. A review of the history, etiology, and clinical findings of craniosynostosis, or premature closure of the cranial sutures, has been presented. The importance of early diagnosis has been stressed in order that mental retardation and cranial deformities may be minimized or prevented. The high incidence of accompanying congenital anomalies elsewhere in the body has been discussed.

2. Two case histories of craniosynostosis, in which successful surgery utilizing polyethylene film was performed, were presented.

A 12-year-old male with scaphocephaly in conjunction with mental retardation and a 14-week-old male with bilateral syndactylism of the hand and possible premature closing of the coronal suture are under observation.

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REFERENCES

1. Ingraham, F. D., and Matson, D. D.: Neurosurgery of infancy and childhood. Charles C Thomas, Springfield, Ill., 1954.
2. Rubin, M. I.: Premature synostosis and associated phenomena, in Practice of pediatrics, edited by J. Brennemann. Chapter 25, vol. 4. W. F. Prior Co., Hagerstown, Md., 1942.
3. Virchow, R.: Cited by Ingraham, F. D., and Matson, D. D., *op. cit.*, ref. 1.
4. Rieping: Cited by Rubin, M. I., *op. cit.*, ref. 3.
5. Park, E. A., and Powers, G. F.: Cited by Rubin, M. I., *op. cit.*, ref. 3.
6. Faber, H. K., and Towne, E. B.: Early craniectomy as preventive measure in oxycephaly and allied conditions with special reference to prevention of blindness. Am. J. M. Sc. 173:701-711, May 1927.
7. Gänslenn, M.: Cited by Rubin, M. I., *op. cit.*, ref. 3.
8. Lane, L. C.: Pioneer craniotomy for relief of mental imbecility due to premature sutural closure and microcephalus. J. Am. M. A. 18: 49, 1892.
9. Ingraham, F. D., Alexander, E., Jr., and Matson, D. D.: Clinical studies in craniosynostosis; analysis of 50 cases and description of method of surgical treatment. Surgery 24:518-541, Sept. 1948.

Problems of Infant Feeding*

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Problems of infant feeding are probably the most discussed in pediatrics today. In reviewing the history of infant feeding,[1] it is interesting to note that for the past 50 years pediatricians and nutritionists have been seeking diligently to improve infant formulas, and it is gratifying to note the resultant progress. At the same time, there has been an increasing insistence on the part of pediatricians and psychiatrists that the maternal breast is by far the best source of infant nourishment and satisfaction—"The milk of every mammal is specific for its young."[2] In the case of human beings, it should not be put aside for either convenience or fashion. This plea for breast feeding dates back as far as the Egypt of the Pharaohs. The history of infant-feeding practices through the centuries, though sketchy, does illustrate rather well, I think, that those infants who survived to adult life were examples of "the survival of the fittest."

In discussing problems of infant feeding, for practical purposes I will divide the infant period into three age levels:

1. Neonatal—first month
2. Infant—1 month to 1 year
3. Transition period—1 year to 2 years

Various feeding problems are peculiar to each period. No apology is offered for the simplicity of material covered, for it is only in reviewing the simple problems that one becomes sharpened for handling the more complex.

By and large, it may be observed that feeding problems per se, which constitute a rather large percentage of the pediatrician's practice, are attributable to adults. The majority of these problems are found in the bottle-fed infant. The emotional and physical forces are closely allied. When physical factors cannot be found as the cause of feeding problems, a search should be made for emotional reactions which, in the modern home, may be evidenced early in infancy.

NEONATAL PERIOD

I will consider two general groupings in the neonatal period which are, in the main, physical causes

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of feeding problems: (1) congenital defects, and (2) nutritional disturbances.

Congenital defects vary considerably in their possible ramifications. It is well to remember that congenital abnormalities are often multiple. They are usually manifested by special symptoms; occasionally they merely interfere with general growth and are discovered only through rather intensive studies of the growth pattern. Almost always, however, gastrointestinal dysfunctions result in disturbed nutritional processes as well as stunted growth.

Obstructive lesions of the gastrointestinal tract due to congenital defects are important causes of neonatal morbidity and mortality.[3] Symptoms and roentgen signs will give adequate identification of these lesions. Early diagnosis provides the best chance for adequate therapy.

The most important sign of intestinal obstruction is vomiting. Obstruction high in the gastrointestinal tract causes early vomiting with minimum abdominal distention. Obstruction proximal to the ampulla of Vater causes bile-free vomitus. Abdominal distention and bilious vomiting are caused by low obstruction; peristaltic waves may be seen through the abdominal wall. In the presence of complete obstruction the rectum may contain quantities of meconium. There are eight obstructive lesions of the gastrointestinal tract:

1. Congenital atresia of the esophagus
2. Congenital hypertrophic pyloric stenosis
3. Obstruction associated with congenital errors in rotation of the midgut
4. Atresia of the intestinal tract
5. Intestinal stenosis
6. Meconium ileus
7. Congenital megacolon
8. Atresia of the anus.

In the neonatal period nutritional disturbances in the breast-fed infant are usually rather mild and are easily corrected. They generally come under the heading of underfeeding or of overfeeding. The symptoms of underfeeding are self-evident: inadequate weight gain or even weight loss and early constipation which later changes to diarrhea in which the stools are slimy, semitransparent, and greenish brown. The behavior of the baby at the breast is typical also, for he will either nurse for long periods (instead of the normal 10 to 15 minutes) and then go to sleep or he will nurse for a few minutes, cry, and then try again. The underfed infant, as time passes, tends to be apathetic and listless, the so-called "too good baby," who nurses poorly with no apparent relish. The weighing of the infant before and after nursing will, of course, give the total 24-hour intake. It must be kept in mind, however, that the amount of milk needed for normal growth may vary and the final criterion is the baby himself.

The treatment of undernourished infants is also self-evident. More food must be obtained, either by mechanical expression of milk from the breast or by supplemental feedings. The mother's diet must be checked, and, almost equally important, an evaluation should be made of her nervousness which is brought on by worry and lack of sleep. Encouraging counsel is needed on both counts. The mother should lead a normal active life. In the case of a first baby, this may be particularly difficult for her. Forced feeding of the mother has no merit, unless it is the mother who wishes to gain the weight. Such is usually not the case. If supplemental feedings are to be offered, they are given after each breast feeding and are of the same composition as for the completely bottle-fed infant. As the infant receives an adequate supply of food, he takes on new vigor, nurses more efficiently, and hence gets more milk from the breast.

I have found it expedient, in those cases in which a young mother wants to nurse her baby and is afraid she cannot do so satisfactorily, to send her home from the hospital with a supplemental feeding formula as a prophylactic procedure. She is to offer the formula to the infant if at any time she is in doubt as to whether or not the infant is satisfied at the breast. Usually, it takes only a few days for the mother to learn that she has an adequate milk supply. In the interim, she has been spared the worries which could have been decidedly detrimental to adequate milk production.

Another cause for underfeeding is aerophagia,[4] and this is more frequently present in the bottle-fed infant. When the infant sucks from a rigid glass bottle, he must release his hold on the nipple after having set up a negative pressure, or he continues to suck and obtains nothing but air. The process of aerophagia may fill the infant's stomach, for physiologically, the presence of air is as effective as food in allaying hunger contractions. This air either escapes slowly or rises to the top and pushes along any food which may have been taken, forcing regurgitation. Brennemann[5] believes that air swallowing is a leading cause of regurgitation during or immediately after feeding in young infants. When this air, added to air already in the stomach, is put under sufficient pressure by ingested food, the only immediate escape route is the esophagus. This problem can be corrected by enlargement of the nipple hole, mechanical devices to prevent bottle vacuum, or the use of a collapsible bottle.

Overfeeding at the breast rarely presents a serious problem. Too much intake of milk at regular intervals will usually correct itself, for the supply will decrease to meet the needs of the infant. Too frequent nursing can be corrected by spacing of feeding periods to not less than 4 hours. "*Vomiting* that occurs immediately after an overabundant nursing is normal, the only *normal vomiting* of infancy."[2] Vomiting which occurs 3 to 4 hours after nursing is usually found in the overfed infant and is due to indigestion. The stool may become greenish, curdy, and irritating to the buttocks, and there may be as many as ten stools a day. An excessive amount of fat in the milk may be the factor, or the infant may have a temporary fat intolerance. Other symptoms of indigestion may be distention of the abdomen with gas, and the discomfort may result in constant crying and loss of sleep. Usually these cases will correct themselves. A teaspoonful of castor oil will often relieve the diarrhea. Occasionally, high protein milk or lactic acid milk may be needed in place of several breast feedings. In these cases I have obtained a certain degree of success by suggesting that 1 or 2 ounces of plain boiled water be offered before each breast feeding.

Colic occurs so commonly in the neonatal period that it is almost to be expected. It is much more frequent in the breast-fed infant, is limited to early infancy, and is found only in the home. That it continues into the early midinfant period is exemplified by the lay term, "3-months' colic."

Authorities are at odds as to the factors causing these agonizing pain paroxysms which cause the infant to scream as he draws his legs up over his abdomen. There are three main theories: (1) hunger and under-

feeding, (2) overfeeding and flatulence, and (3) imbalance of the autonomic nervous system. Brennemann[2] supports the third theory as being the most rational. He feels that infants with immature autonomic systems have gastrointestinal tracts which are not ready for the demands made at birth, either in the matter of normal digestion or of propulsion of gaseous contents. Therefore, in these cases there is an accumulation of excessive amounts of gas in the intestinal tract with resultant distention and then acute obstruction. The excruciating pain which follows is likened to the colicky pain experienced in the adult in such conditions as gas pains following major surgery, peritonitis, intermittent pains of intestinal obstruction or labor, and the most severe pain of gallstones and renal colic.

With this reasoning in mind, the rationale of therapy is understandable. It is important to reassure the mother with whatever optimistic facts about her baby come to the fore in the routine checkup, such as adequate weight gain and growth. The proper technics of nursing and infant care should be reviewed with her, and the need for a calm family atmosphere wherein the baby is not overly handled or disturbed should be stressed. It is important that the baby fit into the household, not the household into the baby.

There are four procedures which I outline for the mother whose infant has paroxysms of colic:

1. Offer the infant warm boiled water by mouth to increase peristalsis.

2. Place the infant over a warm hot-water bottle and apply inhibitive pressure over the sacrum with the heel of the hand.

3. Give the infant a warm soapsuds enema to help gas expulsion. I recommend the use of a number 10 to 12 French urinary catheter as a colon tube for all infant enemas.

4. Give the infant 10 drops of elixir of phenobarbital, and repeat every 20 minutes until relief is obtained. When the child's reaction to phenobarbital is learned, the dosage may be adjusted accordingly. I have found, too, administration of phenobarbital prior to the feeding which precipitates the colic usually helps to break the habit-time element.

Two other familiar procedures are the giving of a casein solution before each breast feeding and the substitution of a low-fat milk, boiled skim milk, or lactic acid milk for an equal number of breast feedings.

In diarrhea of the neonatal period, the character of the stools is of more importance than the number. Direct observation by the physician, too, is of more worth than a description by the mother. I have already mentioned the diarrheal stools which may result from underfeeding and overfeeding. Transitional stools are a normal occurrence between the fourth and seventh day of life. They are loose, greenish yellow or brown and contain mucus. They are the result of gastrointestinal irritation due to the beginning of oral feedings and development of intestinal flora. I mention these transitional stools because of the frantic telephone calls to the doctor which their inception frequently precipitates.

In this period, mild diarrheal disturbances which are due to discrepancies in formula preparation will correct themselves with the substitution of plain water or a solution of 5 per cent glucose for several bottle feedings. In this period in the bottle-fed infant, the more persistent diarrheas (not infectious) necessitate a further evaluation of the formula being used. Boiled skim milk or acidified milk may be substituted with effectiveness.

Constipation during the neonatal period rarely occurs. However, it is well to explain to the mother of a bottle-fed infant that if the baby is eating well and feeling well during this period, it is perfectly normal for him to pass only one stool a day and even at times to miss a day. The mother should be cautioned against the continued use of enemas or suppositories. These measures, when needed for therapy, should be temporary only.

INFANT PERIOD

Allergy and constipation are the only two problems of feeding in the infant period (1 month to 1 year) which I shall consider.

Eczema is the most frequent expression of the allergic state in the first year of life.[6] It is usually considered an atopic manifestation of some specific allergen which has been ingested. Contact dermatitis, resulting from applied medications or, perhaps, proximity to silk or wool, may be superimposed.

The various degrees of infantile eczema and the difficulties which tax the mother and the pediatrician as well as the baby are well known. Before any therapy is started, the physician should adequately reassure the parent that the baby will recover spontaneously at 3 years of age or before, that no scars will result from uncomplicated skin lesions, that the condition is not contagious, and that the baby's health will not suffer because of the eczema. It should be pointed out, too, that remissions of symptoms tend to occur when surroundings are changed, when the seasons change (less prevalent in summer), and, it is hoped, with adequate therapy. Exacerbations are brought about by extreme changes in temperature, ingestion of offending foods and drugs, greasy ointments, contact with certain types of clothing (silk and wool), and by strain and fatigue.

The management of skin manifestations is the most important single factor in therapy. Briefly, treatment includes the use of mineral oil for cleansing, avoidance of soap and water, the use of indicated ointments and lotions for the various lesions, the use of aspirin or phenobarbital to allay itching, and the application of physical restraints to prevent scratching, if necessary. In mild cases, local therapy will usually suffice. Dietary management in the more severe cases is comparatively simple, if not always satisfying, because of the limited foods of which the infant partakes. Orange juice and fish oils should be replaced by crystalline vitamins. Breast milk should not be discontinued unless a thorough investigation has ascertained that something which cannot be removed from the mother's diet is the cause. Hypoallergic milks may be offered in place of formula feedings. New foods may be added as skin manifestations abate. In order to give an adequate trial, not more than one new food should be offered every week.

There are three types of hypoallergic milks and the manufacture of all is based on the precept that the protein molecule is most often the offending allergen.[4] The rationale, then, is to change this molecule.

1. The first group of hypoallergic milks consists of those in which the milk protein is of a species other than cow. The goat is the only other domestic animal available for milk production. Meyenberg evaporated goat milk and Dale dehydrated goat's milk are two of these.

2. The second group is made up of milks in which a vegetable protein factor is substituted. Soybean is the usual source and almond is used in one. These formulas provide a complete food for adequate growth: Allerteen, Mull-Soy, Sobee, Soylac, and Almond-lac.

3. In the third group the protein molecule is changed in structure by heat treatment. This is also known as denaturing the protein. Hypo-Allergic (liquid and powder) and Nutramigen (a casein hydrolysate is used) are examples. Hi-Pro and evaporated milks also alter the protein molecule to a degree in the thermal treatment incident to their manufacture. Foods other than milk which are more readily tolerated by eczematous infants are prune juice for fruit juice, rice or cornmeal for cereal, stewed apples or pears, and mashed ripe bananas.[7] The older infant may be able to tolerate Ry-Krisp instead of bread, well baked potato, well cooked and mashed peas and lima beans, and broiled minced beef or lamb.

It is during the period from 1 month to 1 year of age that a mother occasionally brings her infant to the doctor with the complaint of constipation. The infant is usually bottle fed; repeated enemas, suppositories, or cathartics have been used; and the problem has become very difficult.

The cause of constipation is anatomic, dietary, metabolic, or functional.[5] Whatever the initial cause, by the time the infant is brought to the physician's office the functional aspect has been superimposed and a vicious cycle set up.

An anatomic defect in the intestinal tract which may cause constipation probably has been suspected, if not diagnosed before this period. The causative factor may be an exceedingly long sigmoid colon, redundancy of the transverse colon due to variations in the mesenteric attachments of the bowel, beginning megacolon, or anorectal fissure.

Dietary causes include any condition which leads to a reduction of food intake, for ingestion of food is necessary for the normal stimulation of peristalsis. The qualitative aspects of food intake are important. The relative proportions of milk protein and sugar affect the frequency and consistency of stools. Sugars tend to have a laxative effect; milk proteins, particularly casein, tend to produce constipation. Carbohydrates are inclined to offset the effect of casein because of their effect on the bacterial flora of the bowel.

Constipation of a metabolic nature is not common. Under this category, however, may be listed poor muscular tone with resultant atony of the bowel such as may occur in severe rickets, anemia, and general malnutrition.

The treatment of constipation is directed toward the underlying cause. When inadequate food has been given, an adequate diet should be prescribed. In the standard formula, a change to the more slowly absorbed carbohydrates, such as lactose or maltose, may effect improvements. Failure of response may necessitate reducing the amount of fat in order to avoid production of an excess of calcium soaps (dry, hard gray stools). Fruit juices have a moderate laxative effect (except orange juice). I recommend prune juice to my patients, and usually it is effective, and, occasionally, apricot juice. In those cases in which the habit of nonfunctioning has become the major factor, I at times recommend milk of magnesia to break the habit pattern. Osteopathic manipulative therapy is effective in this type of constipation for it will provide needed stimulation to the intestinal tract for relief of atony and sluggishness.

TRANSITIONAL PERIOD

The problems of the transitional period (1 year to 2 years) which I will discuss are the place of vitamins in an adequate diet and the child who will not eat.

Our knowledge of vitamins has been developed mainly in the last 40 years. Vitamins were first called accessory food factors. It is recognized readily that the lack of any one of the vitamins in the diet results in the development of a deficiency disease. A great deal of work, however, has been done in recent years to learn the minimum daily requirements of the different vitamins for optimum health.[8] It is now known that the amounts of various vitamins necessary to prevent evidence of deficiency disease are entirely different from the amounts required to produce optimum health. An example of this is the fact that 5 to 15 mg. of ascorbic acid will prevent scurvy in the adult, but gingivitis has been noted more frequently on this level than when 65 to 75 mg. is taken daily.

Many foods produced today contain practically no vitamins. White flour and other highly milled products are extensively used because of their cheapness, palatability, and excellent keeping qualities. When all their various forms are included, such as breakfast cereals, rice, macaroni, and bread, they often supply over 30 per cent of the calories consumed. Refined sugar is another widely used product, which, in all its forms, supplies an additional 15 per cent. Thus in the average diet approximately 50 per cent of the calories are supplied by practically nonvitamin foods. From this the importance of vitamin-rich foods in the diet of the infant in the transitional period should be clear.

Vitamins are generally administered as a prophylaxis to the infant who is less than a year old. After this time, the diet is supposed to provide adequate vitamin intake, except perhaps during the winter months, when the addition of vitamins is found to be expedient.

An adequate amount of vitamins, with the exception of vitamin D, can be obtained if the daily diet has as its basis the following six groups of foods: dairy products (milk, cheese, butter), meat, eggs, whole grain or enriched products, vegetables, and fruit; some of the fruits and vegetables should be raw. Any additional calories needed can be obtained from cereals and sugars.

The education of the mother as to what constitutes a balanced diet will materially assist in reaching the aim of preventive pediatrics—optimum health for the child.

It is indeed strange that in spite of the great strides made in infant feeding during recent years, feeding problems of a different nature have developed in older infants, problems which almost offset the progress made.[4] The large percentage of new patients brought to the pediatrician's office with the complaint of not eating is surprising. Much has been written on the subject, many words have been spoken, and zealous manufacturers of vitamins and vitamin-containing foods have recommended many solutions.

These cases are psychogenic rather than organic in origin. Basically, the problem stems from the fact that during the first two periods of infancy, the modern mother tends to measure the intake of her child in terms of those modern measuring units—the weight scale and the magic words "calorie" and "vitamin." Instinct for food taking, quantity, and personal selection are lost in the cloud of enthusiasm for "the knowledge of nutrition."

It should be remembered that this problem is seldom seen in large families or in institutions for the care of children. The difficulty is seen mostly in the home where the number of children is few and the parents have the time to be overly zealous in the care of their offspring.

The problem follows a typical pattern of development which is at first evidenced on the part of the mother when she forces the baby to finish the last ounce of formula prepared or the last teaspoonful of vegetable. When negativism makes its appearance in the baby, about the age of 1 year, these tactics meet with opposition which in time may become quite forceful! Then the picture changes to show a child who schemes to overcome mealtime problems—even to the extent of vomiting food which he has been forced to eat. He has learned the simple technic that refusal pays premiums, and to him this is more pleasant, more rewarding, and the most frequently used method. In order to get the child to eat the parents go through the paces of telling stories and playing all kinds of games, wearing themselves out completely, and amusing the child to no end.

Much of this, if not all, could have been prevented if, in the earlier periods of infancy, the doctor had given instruction stressing the variety of food offered rather than the quantity. The relative importance of milk as an accessory food to the diet, rather than the quantity ingested, is taught by intelligent repetition. This does take time on the part of the busy doctor, but it is more than worth the time spent on re-education lectures which have to be given later when the problem has become fully developed, for, by this time, the mother's mind is pretty much closed to the careless platitude, "If he doesn't want to eat, let him go hungry."

All pediatricians are familiar with the experiment of Davis[9] in which 15 children (6 to 11 months old) over a period of 4½ years successfully chose a balanced diet from a large selection of foods offered three times a day. No physically well or mentally normal child will suffer any clinical degree of malnutrition if a balanced diet is offered to him by the parent with no emotionalism attached. Violation of this axiom is the basis of the anorexia problem in the home of today.

Many of the problems of later childhood have their origin in the failure to solve the noneating habit. When I meet the "he-won't-eat" problem in my office, I ascertain with care the type of parents with whom I am dealing and the factors leading up to the present dilemma. I then evaluate the physical status of the child through a routine physical examination, an erythrocyte count, and hemoglobin determination. When the child's physical status is normal, I can then explain to the mother the technic for overcoming the dilemma.

The placing of balanced meals in small amounts before the child three times a day is simple enough, but the handling of these simple meals requires re-education of the parents. They must understand that food consumption no longer has priority as conversation subject number 1, nor is entertainment to be offered with each bite. The line from the song, "Accentuate the positive and eliminate the negative," is a fairly adequate guide for the handling of these youngsters. The emotional factor of negative response is simply ignored. When the child eats well, he is complimented, but that is the only time that food is mentioned. The typical reactions on the part of the child treated with this new regimen should be outlined by the physician so that from the beginning the mother can recognize progress, thereby encouraging her to stick with the program. The use of tonics and vitamin supplements to increase the appetite must be discouraged. They are not only unnecessary, but they tend to divert parental attention from the real mechanisms underlying the eating difficulty.[10]

Another problem of the transitional period is anorexia which is not psychogenic, but caused by hypoproteinosis.[11] Anorexia is always present in hypoproteinosis, and the familiar symptoms which round out the usual picture are failure to gain weight and to grow normally, irritability, frequent gastrointestinal upsets including vomiting and constipation, repeated bouts of infectious disease, dental cavities, and varying degrees of iron-deficiency anemia. The child's diet is nearly always sufficient in minerals and vitamins but not in protein. Generally, milk and carbohydrate foods are taken in large quantities, but meat, eggs, and other protein foods are neglected. Prompt and dramatic response to provision of adequate protein has established this syndrome as a true deficiency disease.

A dietary history is important in these cases, for usually, according to mother, the child is a good milk drinker but he eats very little at mealtime. Further questioning will reveal that the child does eat between meals—cookies, crackers, and bottled drinks—all highly refined carbohydrate foods. The mother is apt to answer dietary questions with answers which she thinks are correct, rather than by telling the truth. "Does the child eat meat?" "Yes," is the reply she feels is correct, but, to be truthful, often the answer should be, "Only a bite or two, along with lots of potatoes, and he loves his dessert."

Milk has been overemphasized to the general public, particularly because of its calcium content. Milk provides only 1 gram of protein to the ounce; the recommended daily allowance for young children is 40 to 50 grams.

Parents do not realize generally, too, that growth and appetite, rather than energy and appetite, are interdependent. Since growth occurs in varying degrees at various age levels, appetite must vary accordingly. The amount of energy expended by the child has very little to do with the quantity of food that he will consume.

In the management of these cases, too, re-education of the mother is of great importance. An attempt is made to add the word "protein" to the lay vocabulary, alongside the already firmly entrenched "vitamin." A diet adequate in protein is prescribed, and in cases in which large quantities of milk have formerly been consumed, it is better to limit milk consumption to 1 pint or even to omit it entirely from the diet for a short time. The procedures for the mealtime atmosphere which were recommended for psychologic feeding problems are of equal importance in these cases.

As an adjunct to dietary therapy a simple iron preparation is prescribed in these cases because of the varying degree of hypochromic anemia which is a usual accompaniment.

Therapy of hypoproteinosis, as so outlined, is dramatic in its results, but it is impossible to overemphasize the importance of proper education of the parent, by the physician, during the earlier periods of infancy as prophylaxis against the development of this condition.

SUMMARY

The common causes of infant feeding problems during the three periods of infancy have been discussed.

In the neonatal period, consideration has been given to congenital defects and nutritional disturbances.

Allergy, particularly infantile eczema, and constipation in the infant period have been considered.

The role of a vitamin-adequate diet in optimum health and the handling of the child with anorexia have been included in the transitional period.

The importance of proper education of the parents by the pediatrician in avoiding and correcting many infant-feeding problems has been stressed.

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REFERENCES

1. Davidson, W. D.: Brief history of infant feeding. J. Pediat. 43: 74-87, July 1953.
2. Brennemann, J.: Breast feeding and mixed feeding, in Practice of pediatrics, edited by J. Brennemann. Chap. 25, vol. 1. W. F. Prior Co., Hagerstown, Md., 1942.
3. Girdany, B. R., and Sieber, W. K.: Obstructive lesions of intestinal tract in newborn infant. Pediat. Clin. North America, pp. 303-320, May 1954.
4. Meyer, H. F.: Essentials of infant feeding for physicians. Charles C Thomas, Springfield, Ill., 1952.
5. Brennemann, J.: Minor nutritional and digestive disorders of artificially fed infant, in Practice of pediatrics, edited by J. Brennemann, Chap. 27, vol. 1. W. F. Prior Co., Hagerstown, Md., 1942.
6. Nelson, W. E., ed.: Mitchell-Nelson Textbook of pediatrics. Ed. 5. W. B. Saunders Co., Philadelphia, 1950.
7. Andrews, G. C.: Diseases of the skin. Ed. 3. W. B. Saunders Co., Philadelphia, 1946.
8. Tisdall, F. F.: Vitamins, avitaminoses and dietary deficiency diseases, in Practice of pediatrics, edited by J. Brennemann. Chap. 31, vol. 1. W. F. Prior Co., Hagerstown, Md., 1942.
9. Davis, C. M.: Self-selection of diet by newly weaned infants; experimental study. Am. J. Dis. Child. 36:651-679, Oct. 1928.
10. Bakwin, H., and Bakwin, R. M.: Clinical management of behavior disorders in children. W. B. Saunders Co., Philadelphia, 1953.
11. Lynch, H. D., and Snively, W. D., Jr.: Hypoproteinosis of childhood. J. Am. M. A. 147:115-119, Sept. 8, 1951.

The Efficacy of New Drugs in the Treatment of Epilepsy*

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The idiopathic forms of epilepsy submit more readily to therapy than do the symptomatic or organic forms.[1,2] In the presence of organic brain disease, unexpected and contradictory drug reactions may be met. For instance, Tridione can be more effective than Dilantin in the treatment of grand mal epilepsy due to organic disease.[2]

In the treatment of epilepsy it will be found that in most cases combinations of anticonvulsants work better than one drug alone and that many drugs will exert a beneficial effect in many types of epilepsy but are most effective in one type.

When beginning treatment with a single drug, the dose should be gradually increased until the desired results are achieved or toxic symptoms appear. If only partial control is obtained, the dose should be maintained while another drug is added to the regimen. Some patients respond better to one drug than another. Discontinuance of a drug should be gradual in order to prevent the occurrence of status epilepticus. While a drug may control one type of seizure, it can aggravate or precipitate another type if more than one type of epilepsy is present.[2,3] If a drug is to be of any value, its antiepileptic effect should precede its toxic effect.

For purposes of simplicity, drugs will be grouped according to the type of seizure in which they have shown the greatest effectiveness, grand mal, psychomotor attacks, and petit mal.

GRAND MAL EPILEPSY

Among the sedatives which have shown usefulness in the grand mal type of seizures are Gemonil, Mebaral, and Mysoline. These are sedative and anticonvulsant and are believed to protect the unstable brain from the epileptic discharge.

Both Gemonil and Mebaral are similar to phenobarbital in activity but exert a less soporific effect and are relatively nontoxic.[4] They are valuable adjuvant or alternate drugs, used when other drugs may fail to control seizures. Both can be used in place of phenobarbital when it is ineffective or causes excessive drowsiness.

Gemonil seems to show greater effectiveness in organic than idiopathic types of epilepsy, and, if this is corroborated in further studies, it can become a most valuable anticonvulsant.[2] Gemonil appears to be very effective against infantile myoclonic seizures.

Mysoline has been used in those cases which have proved resistant to other drugs and has shown some advantages.[5] When given too rapidly, it causes ataxia and drowsiness. Even when the dosage is built up gradually, patients experience drowsiness, vomiting, and giddiness and, occasionally, hysterical fits.[6] The response, in those cases which have been resistant to other drugs, has been a 50 to 60 per cent improvement with comparatively low toxicity. Therefore, Mysoline shows good promise for future use.[7]

Among the various hydantoin derivatives experimented with, Dilantin sodium and Mesantoin are the most well known, but research is also being done on Hydantal, Thiantoin, Nirvanol, and sodium diphenylene hydantoin. Most members of this group have proved to be highly toxic; Nirvanol is low in activity and high in toxicity. The hydantoins, in general, are said to act by inhibiting the epileptic process directly without depressing cerebral activity.[8,9]

Thiantoin has been known to cause hepatitis, aplastic anemia, and agranulocytosis. Its effectiveness and side actions are similar to those of Dilantin. Because of its toxicity, Thiantoin is unpopular.[10]

Hydantal may produce fewer side effects than Dilantin sodium, but it has caused bone marrow depression. Hydantal and Mesantoin, though not necessarily superior to Dilantin, have demonstrated safety and efficacy in many respects in the treatment of epilepsy.

Another drug which has recently appeared commercially is Hibicon.[11] This drug has shown some merit in partial control of seizures but not enough to be of great value in the treatment of major motor epilepsy.

*Presented at the meeting of Region I, American College of Osteopathic Pediatricians, New York City, March 26, 1954.

Besides, the drug is expensive and has to be used in great dosage.

Themisone, another new anticonvulsant, has been extremely effective in grand mal and psychomotor epilepsy but has produced serious toxic effects, the most serious being aplastic anemia.[4]

PSYCHOMOTOR ATTACKS

Almost all drugs mentioned have some value in psychomotor epilepsy. Mysoline, Mebaral, and Hydantal have a fairly good effect on psychomotor types. Mebaral is said to be very effective in temporal lobe epilepsies, and there are high hopes that this will be proved, since there are very few drugs which have even a slight effect on this type.

Recently Phenurone has attracted a great deal of attention because, next to Dilantin sodium, it has been shown to be the most effective drug for control of psychomotor seizures. Phenurone is an all-purpose drug, being effective in all types of epilepsy and controlling approximately 50 per cent of psychomotor types, 40 per cent of grand mal, and 30 per cent of petit mal.[2] Among its many side effects are hepatitis, bone marrow depression, and induction of psychosis.[12,13] Its efficacy can be enhanced by combining it with phenobarbital and Dilantin.

Phenurone should be used with care, especially in patients who have previously exhibited marked personality problems. It has been noted that when grand mal occurred in combination with psychomotor or petit mal epilepsy, Phenurone exerted greater control than when given, for instance, for grand mal alone. Except in carefully selected cases, Phenurone should not be administered until other accepted anticonvulsants have been tried and found ineffective.[13] In some cases it has brought about an improvement in behavior in patients who already had an existent disorder. Personality disorders need not be a contraindication, but when Phenurone is administered it should be withdrawn if the disorder becomes exaggerated or undesired personality changes occur.

Administration of Phenurone should be highly controlled, even to the extent of supplying only enough of the drug to last for a designated interval.

PETIT MAL EPILEPSY

The "-diones" are still the most popular drugs for treatment of petit mal types of epilepsy. Tridione has been most frequently used and is the most effective. Paradione is similar in general therapeutic action and toxic effects.[3]

Since epilepsy is highly individualistic, some patients experience greater benefit or less troublesome side effects from Paradione, especially in respect to photophobia.[2] Therefore when Tridione fails the analogue may be tried. Paradione has fewer side effects and is more effective in controlling petit mal due to organic brain disease.

Milontin has recently become available for use in petit mal. Milontin has also shown some activity in psychomotor types. Some of its toxic reactions are drowsiness, glomerular and tubular renal damage, rashes, and fever. Milontin, in general, has shown less effectiveness in petit mal than have the "-diones."[14,15]

Recently Prenderol has been investigated in the treatment of petit mal.[2] It has been claimed to be more effective than Paradione and has been shown to control idiopathic petit mal seizures which the "-diones" have been unable to stop. Because it is rapidly absorbed and excreted, it must be given with a retarder such as oil, cream, or carbomethyl cellulose. Prenderol has synergistic action with both "-diones." The need for a retarder has made its use cumbersome. Prenderol can precipitate grand mal seizures even while it controls petit mal attacks.

Finally, there are a number of drugs which have either some anticonvulsant activity or blocking action on some of the side effects of anticonvulsants.

Antihistamines are being used in combination with anticonvulsants, especially in those patients with an allergic background. Central nervous system stimulants such as Benzedrine are being used to diminish the soporific action of anticonvulsants.

SUMMARY AND CONCLUSIONS

An attempt has been made to gather information on new drugs used to control epileptic seizures. Some of the drugs show definite promise for future use. Others are in the experimental stages and are not as yet available for use.

In general, the treatment of epilepsy should start with the drugs most generally in use. Then, if failure is encountered, others should be tried. Besides drug therapy, the management of epilepsy should include proper diagnosis, dietary control, and psychiatric care.

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REFERENCES

1. Alpers, B. J.: Clinical neurology. Ed. 2. F. A. Davis Co., Philadelphia, 1949.
2. Perlstein, M. A.: Drug therapy of epilepsy; with special reference to newer drugs. M. Clin. North America 38:473-484, March 1954.
3. Lennox, W. C.: Control of seizures with drugs. Mod. Med. 19:57-64, March 1, 1951.
4. Conn, H. F., ed.: Epilepsy, in Current Therapy. W. B. Saunders Co., Philadelphia, 1954, pp. 674-681.
5. Doyle, P. J., and Livingston, S.: Use of Mysoline in treatment of epilepsy. J. Pediat. 43:413-416, Oct. 1953.
6. Butter, A. J. M.: Mysoline in treatment of epilepsy. Lancet 264:1024, May 23, 1953.
7. Selby, G.: Mysoline; clinical evaluation of new drug in treatment of refractory cases of epilepsy. M. J. Australia 40:709-715, Nov. 7, 1953.
8. Fabing, H. D., and Hawkins, J. R.: New anticonvulsant drugs in treatment of epilepsy. Ohio State M. J. 44:257-259, March 1948.
9. Brick, H.: Treatment of epilepsy with Mesantoin or Hydantal; study of 35 consecutive patients with grand mal seizures treated at Virginia State Penitentiary and in private practice with follow-up of 1 to 2 years. Virginia Med. Month. 78:423-429, Aug. 1951.
10. Van Middlesworth, L., and Kyle, J. W.: Acute hepatic insufficiency occurring during therapy with phethenylate (Thiantoin) sodium. South. M. J. 45:1180-1181, Dec. 1952.
11. Livingston, S.: Use of Hibicon in treatment of epilepsy. J. Pediat. 43:673-674, Dec. 1953.
12. Tyler, M. W., and King, E. Q.: Phenacemide in treatment of epilepsy. J. Am. M. A. 147:17-21, Sept. 1, 1951.
13. Keith, H. M.: Use of Phenurone in convulsive disorders in children. Am. J. Dis. Child. 80:719-724, Nov. 1950.
14. Doyle, P. J., Livingston, S., and Pearson, P. H.: Use of Milontin in treatment of petit mal epilepsy; three per second spike and wave dysrhythmia. J. Pediat. 43:164-166, Aug. 1953.
15. Millichap, J. G.: Milontin; new drug in treatment of petit mal. Lancet 2:907-910, Nov. 8, 1952.

ADDITIONAL REFERENCES

Chen, G., and Ensor, C. R.: Evaluation of antiepileptic drugs. Arch. Neurol. & Psychiat. 63:56-60, Jan. 1950.

Conn, H. F., ed.: Epilepsy, in Current therapy. W. B. Saunders Co., Philadelphia, 1953, pp. 619-624.

Mackay, R. P.: Epilepsies; their diagnosis and treatment. J. Michigan M. Soc. 52:618-625, June 1953.

Pearlstein, M. A.: Gemonal (5, 5-diethyl 1-methyl barbituric acid); new drug for convulsive and related disorders. Pediatrics 5:448-451, March 1950.

Whitty, C. M. M.: Value of Primidone in epilepsy. Brit. M. J. 2: 540-541, Sept. 5, 1953.

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Immunization in Pediatrics*

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The pediatrician must know how to utilize the vaccines and serums which immunologists have developed for the prevention of many important diseases. These obviously play an important role in the protection of the community.

There is probably no greater accomplishment in preventive medicine than that of active immunization.

PRENATAL IMMUNIZATION

Before discussing active immunization, prenatal multiple immunization should first be considered. Whooping cough and diphtheria are still the two most important preventable diseases of infancy. About 50 per cent of mothers, and therefore their offspring, are not immune to diphtheria, and only 15 to 30 per cent are immune to pertussis.[1] In agglutination titers taken immediately after delivery from women immunized against pertussis in the last trimester of pregnancy and from their offspring (using cord blood), 84 per cent showed satisfactory titers or better (1:300 being considered the minimal level for good immunity). Those infants who had a titer of 1:500 or over at birth, approximately 35 per cent of the total observed, were retested at the age of 3 months, and good immunity was retained by 44 per cent. At 6 months, 25 per cent of this group were retested, and in no instance did a demonstrable agglutination titer appear.

When the blood was examined for circulating diphtheria antitoxin, 61 per cent showed adequate immunity to diphtheria at 3 months and 12 per cent at 6 months, but no immunity was evidenced at 9 months.[1]

As with pertussis, passive immunity against diphtheria through prenatal immunization appeared to wane rapidly after the age of 3 months. The response of pregnant women to one injection of influenza vaccine was similar to their response to pertussis and diphtheria immunization.

Thus it must be concluded that multiple immunization of the expectant mother would seem to protect the newborn against diphtheria, pertussis, influenza, and, possibly, tetanus during the first 3 months of life, when effective active immunization cannot be established. Active immunization should follow at the age of 3 to 4 months, when passive immunity has been lost and mechanisms for establishment of active immunity function more efficiently.[1]

DIPHTHERIA, TETANUS, AND PERTUSSIS

Little purpose would be served here by reviewing the history and development of the immunizing agents now commonly employed; it is sufficient to say that these produce supposedly satisfactory levels of immunity in most infants and that responses are probably adequate when injections are given early in infancy.

The use of the familiar combinations, usually triple-antigenic materials such as combined alum-precipitated or aluminum hydroxide-adsorbed diphtheria and tetanus toxoids containing Hemophilus pertussis organisms, is considered preferable to antigens suspended or dissolved in plain diluents.[2] The reasons for this are:

1. More prolonged antitoxic immunity
2. Much greater effectiveness as immunizers against pertussis in early infancy
3. Less likelihood of producing systemic reactions because of the lower protein content and slower absorption, even though the combined fluid mixtures (nonadsorbed) have the advantages of (1) greater speed in achieving immunization and (2) freedom from occasional sterile abscess formation.

Precautions.—

1. Autoclaved needles and syringes are preferable, or equipment should be boiled for 10 minutes at least.
2. The site of inoculation as well as the rubber stopper of the antigen vial should be sterilized.
3. Antigens, alum-precipitated or aluminum hydroxide-adsorbed vaccines, should be injected intramuscularly; fat necrosis may be avoided by terminating the injection of 0.1 cc. to 0.2 cc. of air.
4. Fluid antigens without adjuvants, that is, fluid toxoids and saline-suspended vaccines, should be given subcutaneously.
5. Only well infants should be injected.
6. Acetylsalicylic acid, 1 grain per year of age, should be given within 1 or 2 hours of each injection and repeated in 4 hours.
7. Infants with histories of febrile convulsions should be injected with fractional doses of antigen, beginning with 0.1 cc.
8. When an infant is brought in for his second injection the parent should be questioned about the occurrence of fever, somnolence, and local reaction following the previous injection. If these are reported, the volume of the next injection should be appropriately decreased. If a convulsion or a severe reaction is reported, no further injections should be given for a year.

Contraindications.—

1. Any respiratory involvement or other acute infection is reason for delaying injections. Prolonging the interval between injections, even up to 6 months, rarely, if ever, interferes with the final immunity.
2. The presence of cerebral damage in the infant is an indication for delay in immunization. Severe febrile reactions, with or without convulsions, may be encountered. In such infants, active immunization procedures should not be started until after they are 1 year of age. Fractional doses should be employed to test the infant's tolerance before full doses are used.
3. The occurrence of an outbreak of poliomyelitis in a community is a signal for deferring elective immunization procedures in individuals over the age of 6 months. Primary immunizations and/or recall injections obviously cease to be elective procedures when outbreaks of diphtheria, pertussis, typhoid, or smallpox coexist with that of poliomyelitis.[3]
4. Infants with eczema, or other forms of dermatitis, should not be vaccinated against smallpox because of the danger of producing generalized vaccinia.
5. Siblings of infants with eczema dermatitis should not be vaccinated against smallpox because of the danger of cross infection. Eczema vaccinatum may be produced by direct skin contact or by droplet infection from the nasopharynx, the secretions of which

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contain virus on the fourth and fifth days after vaccination.

Procedure: Combined Immunization with Adjuvant.—

1. Starting at 3 months of age, 0.5 cc. should be injected monthly intramuscularly with a minimum of three injections. A different site for injection should be used each time, such as for the first injection the left gluteus maximus; for the second, the right gluteus maximus; and for the third, the left vastus lateralis or right deltoideus. If the injections are started later in infancy, then the deltoideus and triceps should be injected. During the course of primary immunization the same muscle should not be injected more than once. If the immunizations are started earlier, there should be four injections in order to administer a minimum of 60 billion Hemophilus pertussis organisms. The presence of an epidemic of pertussis may be an indication for such early combined immunization.

In children not immunized during infancy, combined alum-precipitated or aluminum hydroxide-adsorbed vaccine is still the product of choice. The same dosage schedule should be employed using the deltoideus and triceps. If the child has not yet received his smallpox vaccination, that should be done first and the immunization injections should be started 2 weeks later. Diphtheria toxoid should not be administered without a preliminary Schick test if the child is past the age of 6 or without preliminary testing for toxoid sensitivity if he is past the age of 10. Pertussis vaccine should not be employed if the patient has had whooping cough.

ROUTINE RECALL INJECTIONS (BOOSTERS)

When primary immunization has been performed in infancy using alum-precipitated or aluminum-hydroxide combined preparations, the following procedure is followed for stimulating injections:[3]

One year after completion of the primary course, one full dose of 0.5 cc. is given intramuscularly.

Two years later (about 3 to 4 years of age) three-fifths of a dose (0.3 cc.) is administered intramuscularly.

Every 3 years thereafter recall injections of three-fifths of a dose (0.3 cc.) should be given. These will be given at 6 to 7 years of age and at 9 to 10 years of age.

After 10 years of age, recall injections for pertussis need not be repeated if a primary course and four recall injections have been administered. However, tetanus toxoid recall (booster) injections should be given every 3 years throughout life in 0.1 cc. to 0.2 cc. doses.

A Schick test should be done at the time of every recall tetanus toxoid injection. If needed, diphtheria toxoid, alum-precipitated, is given in a 0.25 cc. dose.

On exposure, recall injections of 0.5 cc. of fluid toxoid should be given subcutaneously for the reasons previously mentioned.

SMALLPOX

Smallpox vaccination is performed either before the combined immunization procedure or soon thereafter. It should always be done before the end of the first year.

Revaccination is recommended every 5 years, and before that time in the presence of exposure to the disease or an epidemic.[4]

BCG

This vaccine is not accepted thus far as a routine measure and is advised only in certain special circumstances and where tuberculosis is widespread, as on Indian reservations. It is not available commercially.

It is indicated for tuberculin-negative children or infants who have contact with a known case of tuberculosis and where separation is impossible.

It must be stressed that BCG vaccination of infants and children should never become a routine substitute for hospitalization of adults with open cases of tuberculosis.

TYPHOID FEVER

Typhoid vaccine is advised for infants and children living in endemic areas, or when travel to such areas is contemplated, and for contacts. Combination vaccine containing paratyphoid A and B organisms is preferred. The injections are subcutaneous and are given at weekly intervals; a recall or booster dose is given every 1 to 2 years depending on the probability of exposure. It is injected intracutaneously in doses of 0.1 cc. The following age-dosage schedule, based on the total adult dosage of 2.5 cc., may serve as a sample:

In infancy one-fifth of the total adult dose, or 0.5 cc., is given: First dose, 0.1 cc.; second dose, 0.2 cc.; and third dose, 0.2 cc.

From 1 to 4 years of age the dosage is two-fifths of the total adult dose, that is, 1 cc.: 0.2 cc. is given at the first injection, 0.4 cc. at the second, and 0.4 cc. at the final injection.

From 5 to 10 years of age, three-fifths of the total adult dose or 1.5 cc. is given in 3 doses of 0.5 cc.

After 11 years of age the adult dosage is used.

ROCKY MOUNTAIN SPOTTED FEVER[5,6]

Immunization for Rocky Mountain spotted fever is recommended in areas where ticks may be infected.

In addition to preventive hygiene, two vaccines are available for protection against the disease. There is possibility of development of a repellant or tick-killing chemical for impregnating clothing.

A vaccine made from the infected yolk sac of the chicken embryo is administered in three subcutaneous injections of 1 cc. each at 5- to 7-day intervals. Protection is better in children than adults and reactions are rare, but severe ones may occur. This type of vaccine has been in use for about 10 years.

Vaccine prepared from infected ticks is distributed to physicians, upon request, for the immunization of those persons who are sensitive to egg protein. It is injected subcutaneously three different times at intervals of 5 to 7 days. This vaccine has been in use for nearly 25 years.

Both vaccines are of equal value and are now manufactured by the Public Health Service. The maximum degree of protection is less than 1 year, hence revaccination yearly is necessary. After this has been repeated each spring for 3 uninterrupted years, injections every 2 years should suffice.

GERMAN MEASLES

There is no active immunization for this disease. Passive immunization is available but is recommended only for the protection of exposed susceptible pregnant women. Actually, the use of gamma globulin for this disease is of questionable value, but the potential danger of rubella to the fetus warrants it. If exposure to German measles occurs during the first 4 months of pregnancy, the suggested dose of gamma globulin is 0.1 cc. per pound of body weight. It is injected intramuscularly.

MEASLES

Passive immunization in measles is feasible with the use of gamma globulin. Gamma globulin will either

modify or prevent the disease if administered shortly after exposure. In most cases, modification of measles is preferred, owing to the seriousness of the disease and in order to produce natural immunity.

It is recommended for prevention in infants from 6 to 18 months of age; infants under 6 months of age whose mothers have not had measles; possibly in hospitals to abort an epidemic; in children of any age who are suffering from a chronic or debilitating illness or operation, severe acute illness, or when another communicable disease is likely to occur at approximately the same time from which gamma globulin will not afford protection.

The recommended dosage for prevention is 0.1 cc. per pound of body weight, given intramuscularly. Modification of measles in all other children between the age of 18 months and 3 years of age who have never had the disease and have been exposed may be accomplished with the use of 0.02 cc. per pound of body weight given during the first 5 days of exposure. If after the fifth day of exposure, the recommended dosage is 0.05 cc. per pound.

INFECTIOUS HEPATITIS

Gamma globulin is effective for passive immunization in infectious hepatitis if given in the recommended dosage of 0.02 cc. per pound of body weight, with a maximum dose of 2 cc.

MUMPS

Active immunization in mumps is not advised except under unusual circumstances, in which case 1 cc. to 2 cc. of inactivated or attenuated virus vaccine from infected chick allantoic fluid is administered subcutaneously. Protection afforded may be from 3 to 12 months.

SUMMARY

The role of the pediatrician in immunization and, thereby, in preventive medicine has been mentioned. Consideration has been given to the results of prenatal multiple immunization. The combined immunization procedure for diphtheria, tetanus, and pertussis, precautions and contraindications, and booster injections have been discussed. Immunization procedures for smallpox, tuberculosis, typhoid fever, Rocky Mountain spotted fever, German measles, measles, infectious hepatitis, and mumps have also been referred to.

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REFERENCES

1. Cohen, P., Schneck, H., and Dubow, E.: Prenatal multiple immunization J. Pediat. 38:696-704, June 1951.

2. Sako, W. S.: Whooping cough, in Communicable diseases, edited by R. L. Pullen. Lea & Febiger, Philadelphia, 1950, pp. 364-375.

3. Platou, R. V., and Horan, J. M.: Pertussis. Bull. Tulane M. Facul. 12:91-98, May 1953.

4. Shaffer, T. E.: Preventive pediatrics, in Textbook of pediatrics, edited by W. E. Nelson. Ed. 6. W. B. Saunders Co., Philadelphia, 1954, pp. 128-138.

5. Report of Committee on Therapeutic Procedures for Acute Infectious Diseases and on Biologicals of American Academy of Pediatrics. American Academy of Pediatrics, Evanston, Ill., 1947.

6. Parker, R. R., and Oliphant, J. W.: Rocky Mountain spotted fever, in Communicable diseases, edited by R. L. Pullen. Lea & Febiger, Philadelphia, 1950, pp. 719-752.

BOOK NOTICES

(Continued)

ESSENTIALS OF PEDIATRICS. By Philip C. Jeans, A.B., M.D., Late Professor of Pediatrics, State University of Iowa, Iowa City; F. Howell Wright, B.S., M.D., Professor of Pediatrics, University of Chicago; and Florence G. Blake, R.N., M.A., Associate Professor of Nursing Education (Nursing Care of Children), University of Chicago. Cloth. Pp. 808, with illustrations. Price $4.75. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

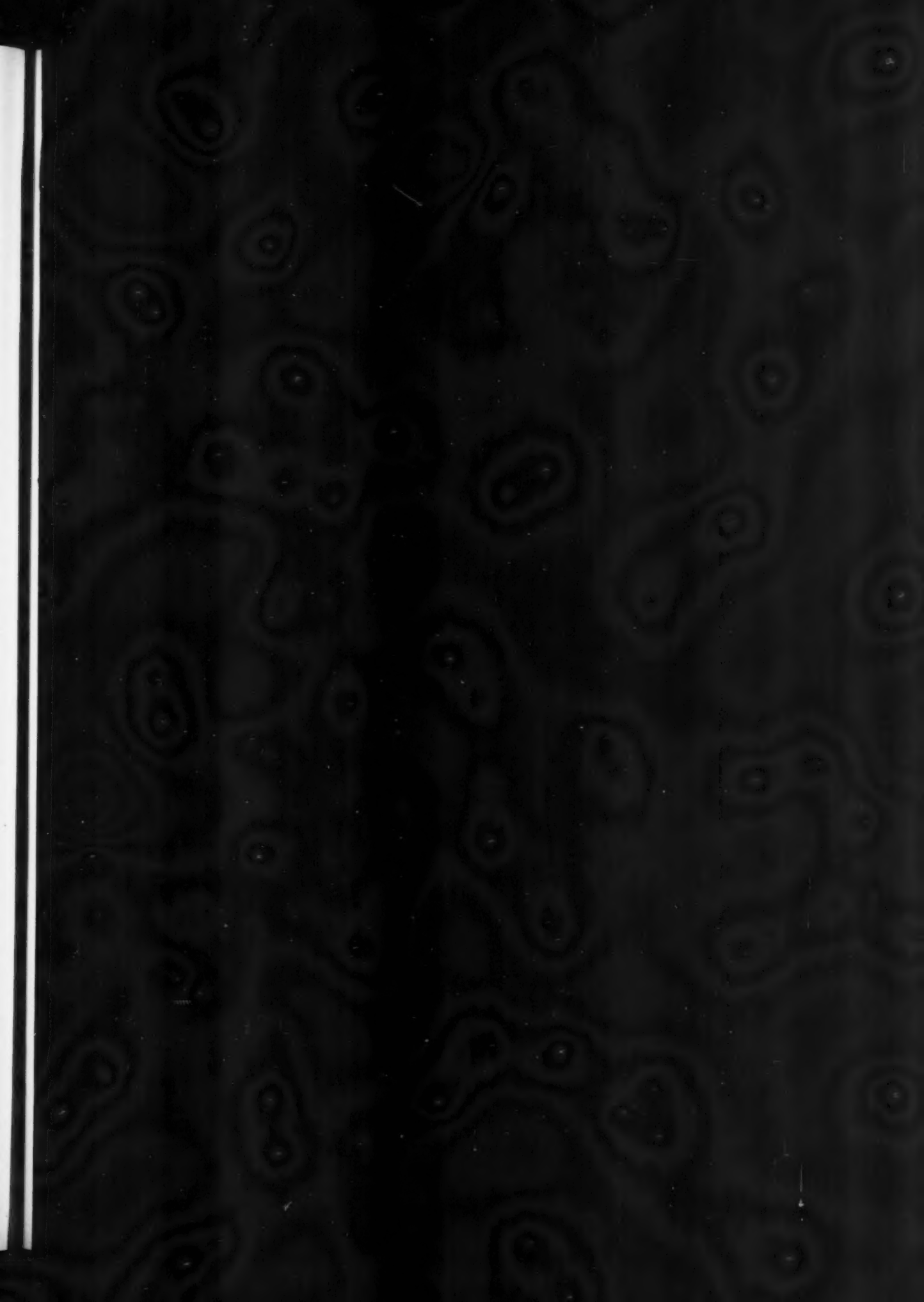
"A nurse should not be called upon to diagnose the nature of an illness and prescribe for a patient, yet a knowledge of these fields is an aid in understanding the reasons for those things which she is required to do," it was stated in the preface to the first edition of this book. Two decades and five editions later, this is still the guiding principle of this widely used textbook on pediatric nursing. The book is organized under five units: orientation, growth, development, care and guidance of the infant and the child; general nursing care; nursing in the care of the sick infant and child; and nutrition and nutritional diseases. The fifth edition has been completely revised within this framework to include a new chapter on the general principles of pediatric therapy, new material on the child's psychologic development from birth through adolescence, a rewritten chapter on disorders of the newborn, modern and up-to-date information on nutrition, rearranged material on the diseases of the nervous system and on the infectious diseases. Dr. Jeans, one of the three authors, died while the book was being revised but not before he had completed a revision of the section on nutrition and had indicated the areas in which modernization was desirable.

A compact text with double-set columns, this book is opened with a detailed table of contents and closed with an equally complete index, which takes up sixty-seven pages. For study or reference, the student or practicing nurse will find this a helpful book not only for discussions on the technics of nursing but also for a comprehensive understanding of the field of pediatrics.

ILLUSTRATED HANDBOOK OF CHILD CARE. From Birth to Six Years. Compiled and illustrated by Wava McCullough; assisted by Marcella Gawronski, R.N., Instructor and Coordinator of Community Nursing, Pasadena City College . . . Huntington Memorial Hospital School of Nursing. Cloth. Pp. 232, with illustrations. Price $4.00. McGraw-Hill Book Company, 330 W. 42nd St., New York 36, 1954.

A visual delight as well as a practical and informative guide on child care is this pictorial handbook. Between the first drawing showing a cheerful nurse recording a baby's footprint and the last illustration of an impish baby waving "bye-bye," the reader will be entertained by the clever artwork and informed by the clear and concise text, both of which are the handiwork of Author McCullough, a practical nurse. The handlettered text and the expressive line drawings, all reproduced in sepia, are in keeping with the book's informality and charm, which are outstanding among the many assets of the book.

The nine chapters, each introduced by an amusing drawing, deal with furnishings, feeding, bathing, training, the play area, the baby sitter, special problems, the sick baby and child, and prevention and first aid. Within this outline are presented helpful, sound suggestions on providing for the mental and physical health and welfare of a child from birth to age six. In imparting the advice, the author reveals her patient understanding of children, which is the result of her many years of experience in caring for babies and children. Her chapters on caring for a sick or hurt child are especially helpful. And parents will be grateful for the chart of childhood diseases—chicken pox, diphtheria, measles, mumps, poliomyelitis, scarlet fever, and whooping cough—that tersely informs how to avoid, how to recognize, and what to do about them. As special problems are discussed such subjects as naming the baby, traveling with children, and how to bring up twins. In short, the author has overlooked nothing on the subject of child care. It is therefore obvious that this is a book that belongs on the reference shelf of all parents.



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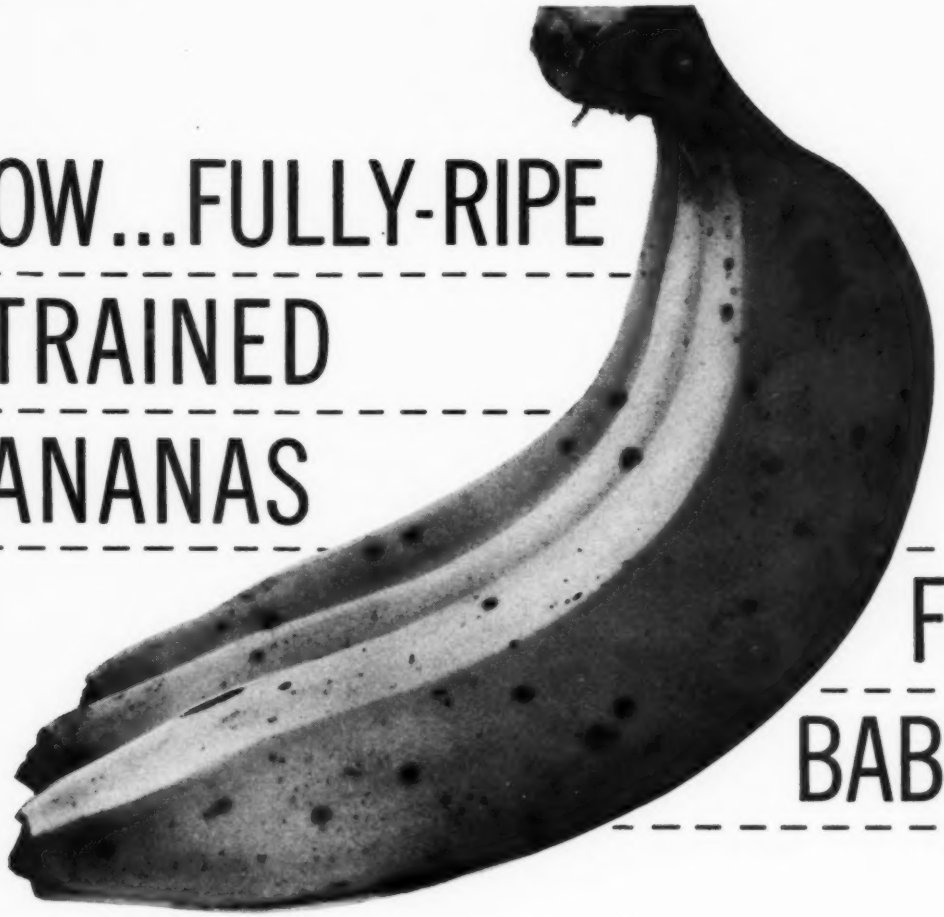

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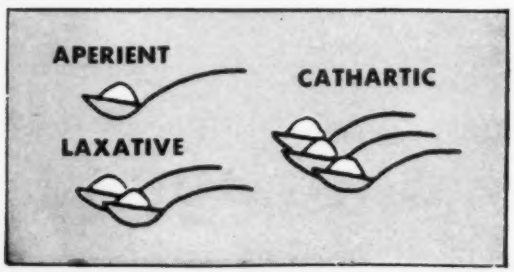
References: 1. The Physiological Basis of Medical Practice, 1945, p. 486. 2. New England J. Med. 235:80, July 18, 1946.



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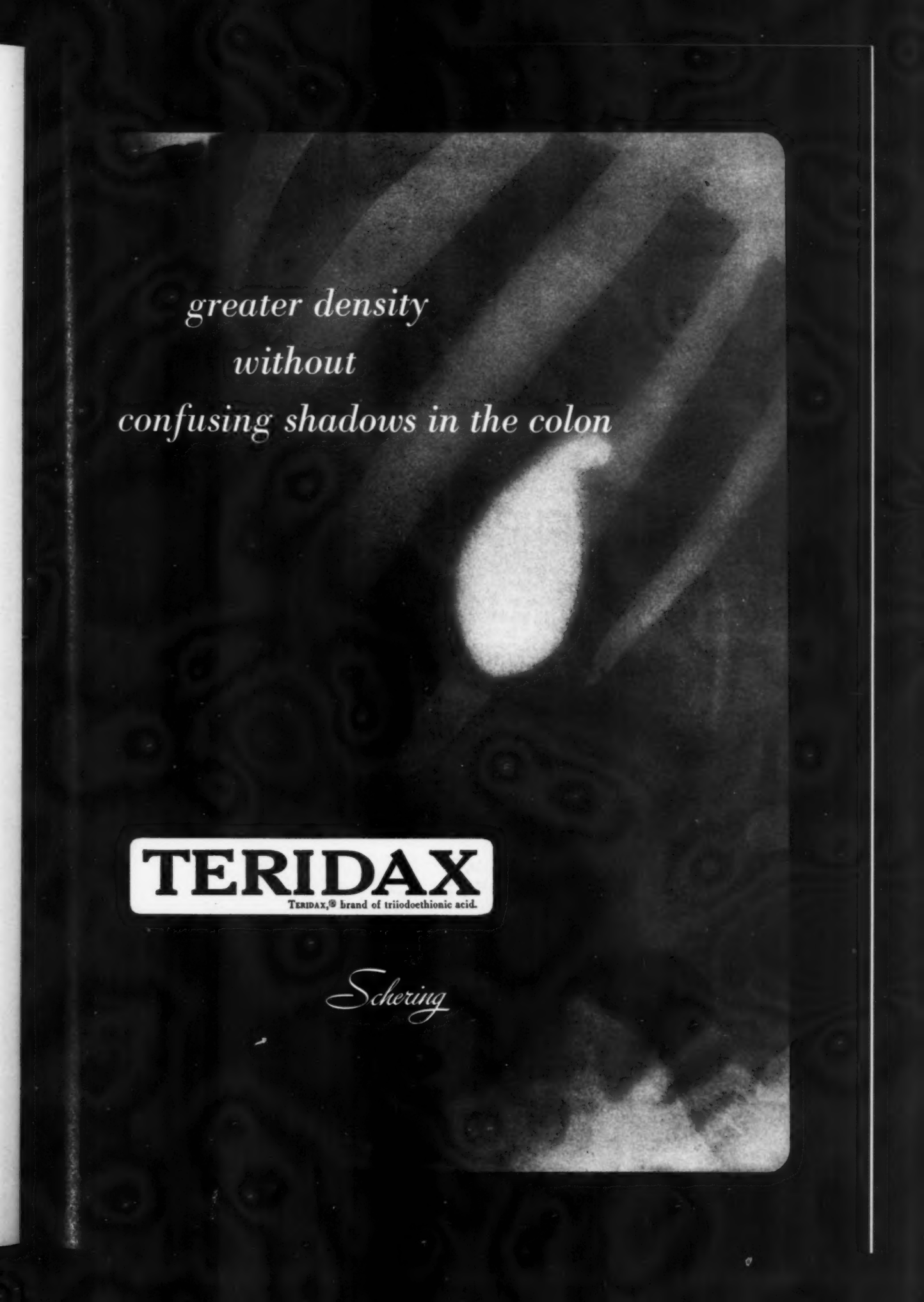
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1. Kloha, M. W.; Draper, M. D., and Keller, F.: J. Am. Chem. Soc. *76*:2843 (May 20) 1954.

2. Cronheim, G.; Brown, W.; Cawthorne, J.; Toekes, M. I., and Ungari, J.: Proc. Soc. Exper. Biol. & Med. *86*:110 (May) 1954.

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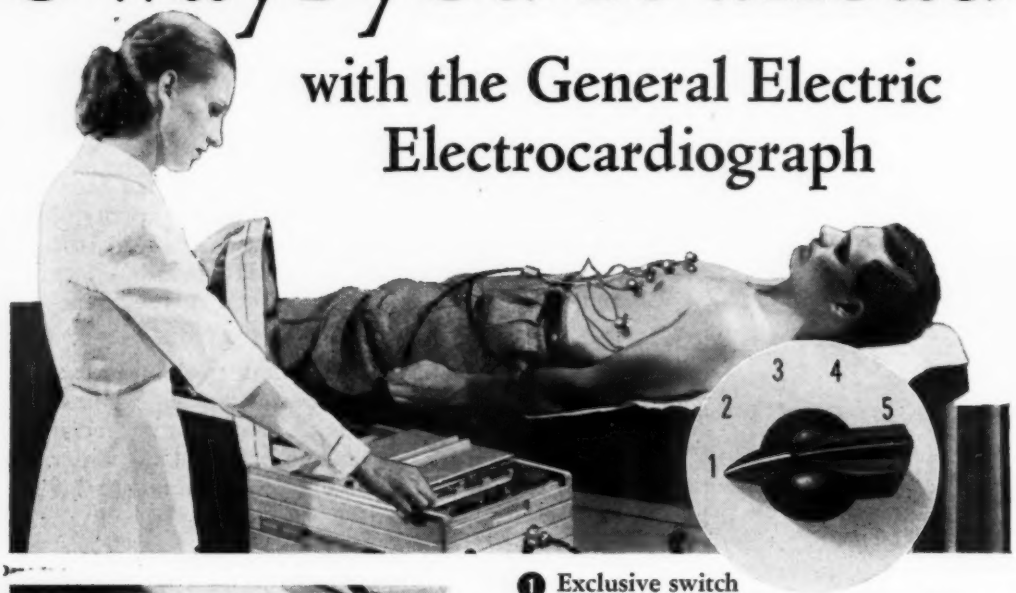

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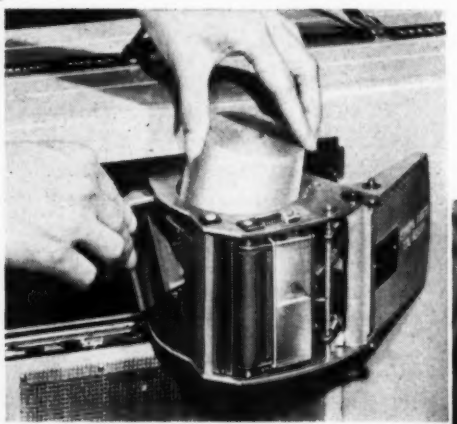

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Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Ninth Annual Convention, Biltmore and Statler Hotels, Los Angeles, July 18-22. Program Chairman, W. Donald Baker, 3450 W. 43rd St., Los Angeles.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Roosevelt Hotel, Jacksonville, Fla., February 7-10. Secretary, Harold K. Morgan, 3268 W. 32nd Ave., Denver 11.

American Osteopathic Society of Proctology, annual clinical assembly, Bellevue-Stratford Hotel, Philadelphia, April 14-16. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif. Program Chairman, Joseph W. Kenney, 718 Guaranty Bldg., Indianapolis.

Arizona, annual meeting, Paradise Inn, Phoenix, April 22-24. Secretary, H. E. Allshouse, 2243 N. 12th St., Phoenix 22.

Eastern Osteopathic Association, annual meeting, Hotel Statler, New York City, April 2-3. Secretary, Frank B. Tompkins, Baltimore Life Bldg., Baltimore 1. Program Chairman, Chester D. Losee, 212 Prospect St., Westfield, N. J.

Georgia, annual meeting, Bon Air Hotel, Augusta, May 6-7. Secretary, C. A. Means, Anderson Bldg., Marietta. Program Chairman, M. Lillian Bell, 2075 Ridgewood Drive, N. E., Atlanta 6.

Indiana, annual meeting, French Lick Springs Hotel, French Lick Springs, May 14-17. Secretary, Arabelle B. Wolf, 809-13 Odd Fellows Bldg., Indianapolis 4. Program Chairman, Paul van B. Allen, 1500 N. Delaware St., Indianapolis 2.

Massachusetts, annual meeting, Hotel Samoset, Boston, January 15-16. Executive Secretary, Mrs. Gladys M. Stockdale, 524 California St., Newtonville 60. Program Chairman, Raymond W. Boyd, 430 Broadway, Lynn.

New Mexico, annual meeting, Albuquerque, April 28-30. Secretary, Robert E. Smith, 205 N. First St., Lovington.

Northwest Osteopathic Convention, annual meeting, Portland, Ore., June 13-16. Program Chairman, Russell F. Kenaga, Sr., 2265 W. Burnside St., Portland 5.

Ohio, annual meeting, Neil House, Columbus, May 2-4. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15.

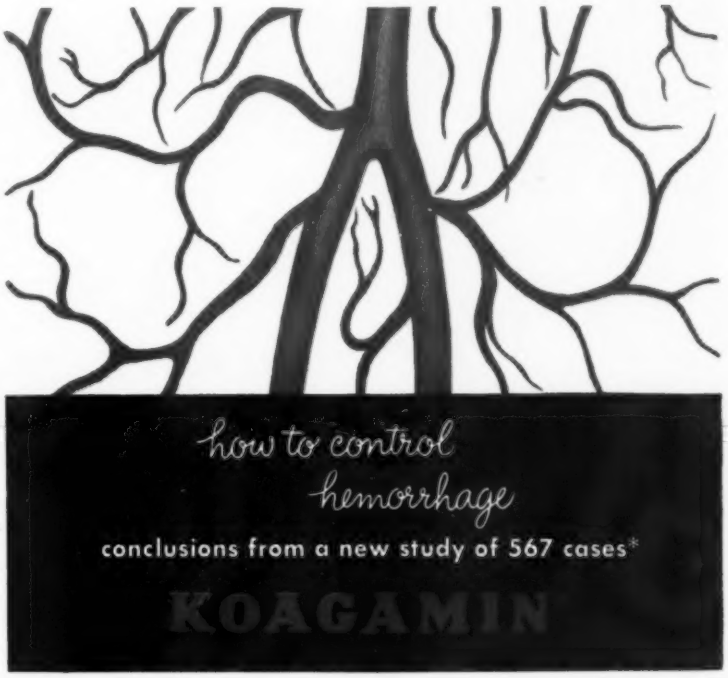
Oregon: See Northwest Osteopathic Convention.

South Dakota, annual meeting, Sylvan Lake Hotel, Black Hills, June 5-7. Secretary, E. W. Hewlett, 417 W. 27th St., Sioux Falls. Program Chairman, Laurence S. Betts, Box 205, Huron.

Utah and Wyoming, joint annual meeting, Logan, Utah, June 26-27. Secretary, Utah Society of Osteopathic Physicians and Surgeons, Alice E. Houghton, 600 Zion's Savings Bank Bldg., Salt Lake City 1. Secretary, Wyoming Association of Osteopathic Physicians and Surgeons, G. A. Roulston, 2823 Central St., Cheyenne.

West Virginia, annual meeting, McClure Hotel, Wheeling, June 4-7. Secretary, Guy E. Morris, 542 Empire Bank Bldg., Clarksburg. Program Chairman, Joseph B. C. Bartram, Gilmer Clinic, Post Office Bldg., Glenville.

Wyoming: See Utah.



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*Joseph, M.: Am. J. Surg. 87:905, 1954.

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ARIZONA

Those interested in professional examinations should contact Russell Peterson, D.O., secretary, Board of Registration and Examination in Medicine and Surgery, 2747 East McDowell Road, Phoenix.

COLORADO

Basic science examinations March 2-3 in the Lecture Room, second floor, Y.M.C.A. Bldg., E. 16th Ave. and Lincoln St., Denver. Applications must be filed by February 16. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver 18.

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References: 1. Lee, R. I.: Chicago M. Soc. Bull.: 48:503, 1946. 2. Golob, M.: Am. J. Digest. Dis. 18:308, 1951. 3. McLester, J. S., and Darby, W. J.: Nutrition and Diet in Health and Disease, ed. 6, Philadelphia, W. B. Saunders Company, 1952, pp. 416, 476.

CONNECTICUT

Basic science examinations February 12 at New Haven. Applications must be filed 2 weeks prior to examination. Address Mr. Herbert S. Harned, secretary, Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DELAWARE

Examinations January 11-13. Address Joseph S. McDaniel, M.D., secretary, Board of Medical Examiners, 229 S. State St., Dover.

IDAHO

Susan B. Kerr, McCall, and E. J. Miller, Nampa, have been appointed to the Board of Osteopathic Examiners for terms expiring in April, 1956.

ILLINOIS

Examinations January 26-28, 160 N. LaSalle St., Chicago. Address Mr. Frederic B. Selcke, Superintendent of Registration, Department of Registration and Education, State House, Springfield.

IOWA

Basic science examinations January 11 at the Capitol Bldg., Des Moines. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

Examinations February 17-19 at Topeka. Address Forrest H. Kendall, D.O., secretary, Board of Osteopathic Examination and Registration, 420½ Pennsylvania, Holton.

MASSACHUSETTS

Examinations January 11. Address Robert C. Cochrane, M.D., secretary, Board of Registration in Medicine, Room 33, State House, Boston 33.

MICHIGAN

Basic science examinations February 12-13 at Detroit and Ann Arbor. Applications must be filed prior to February 1. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, Mason Bldg., Lansing.

MONTANA

Examinations March 1. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic science examinations January 11-12. Address Mr. Husted K. Watson, Director, Bureau of Examining Boards, Department of Health, State Capitol Bldg., Lincoln 9.

NEVADA

Professional examinations January 29 at 210 W. Second St., Reno. Applications must be filed by January 15. Address Walter J. Walker, D.O., secretary, Board of Osteopathic Examiners, 210 W. Second St., Reno.

Basis science examinations January 4. Address Donald G. Cooney, Ph.D., secretary, Board of Examiners in the Basic Sciences, Box 9005, University Station, Reno.

NEW HAMPSHIRE

Examinations March 9-10 at Concord. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, State House, Concord.

NEW MEXICO

Basic science examinations January 16. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P. O. Box 1522, Santa Fe.

Lawrence C. Boatman, D.O., Santa Fe, has been reappointed to the Board for a term expiring October 16, 1958.

NEW YORK

Examinations February 15-18. Applications must be filed not less than 30 days prior to examination. Address Dr. John W. Paige, Chief, Bureau of Professional Examinations and Registrations, 23 S. Pearl St., Albany 7.

NORTH DAKOTA

Examinations January 8 at Minot. Applications must be filed by December 15. Address G. L. Hamilton, D.O., secretary, Board of Osteopathic Examiners, Box 1088, Minot.

OREGON

Basic science examinations March 5. Address Mr. John R. Richards, secretary, Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations January 6-7. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

Basic science examinations in Room 366, State Office Bldg., Providence, February 9. Applications must be filed approximately 20 days prior to examination. Address Mr. Casey.

SOUTH DAKOTA

Examinations January 18-19 at Sioux Falls. Address Mr. John C. Foster, executive secretary, Board of Medical and Osteopathic Examiners, Room 300, First National Bank Bldg., Sioux Falls.

TENNESSEE

Examinations in February and July at Nashville. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic Physicians, 1226 Highland, Jackson.

VERMONT

Examinations January 26-27 at the State House, Montpelier. Applications must be filed by January 10. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

WASHINGTON

Professional examinations in the Auditorium of Health Science Bldg., University of Washington, Seattle, January 10-12. Applications must be filed 15 days before examination. Address Mr. Edward C. Dohm, secretary, Professional Division, Department of Licenses, Olympia.

Basic science examinations in the Auditorium of Health Science Bldg., University of Washington, Seattle, January 5-6. Applications must be filed 15 days before examination. Address Mr. Dohm.

WISCONSIN

Professional examinations January 11-12 at Madison. Address Thomas W. Tormey, Jr., M.D., State Office Bldg., 1 W. Wilson St., Madison.

Basic science examinations March 12 at Hotel Loraine, Madison. Applications must be filed by March 4. Address Prof. William H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom Ave., Ripon.

WYOMING

Examinations February 7 at Cheyenne. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

December 31—Tennessee, $5.00. Address M. E. Coy, D.O., secretary, Board of Examination and Registration for Osteopathic physicians, 1226 Highland, Jackson.

Prior to January 1—Arizona, not more than $10.00. Address Russell Peterson, D.O., secretary, Osteopathic Board of Registration and Examination in Medicine and Surgery, 2747 E. McDowell Rd., Phoenix 22.

January 1—California, $20.00 for residents and nonresidents. Address Glen D. Cayler, D.O., secretary, Board of Osteopathic Examiners, 1013 Forum Bldg., Sacramento 14.

January 1—Florida, $5.00. Address R. Philip Coker, D.O., secretary, Board of Osteopathic Medical Examiners, Box 66, Panama City. Doctors who hold Florida licenses and who have moved since last year should advise the secretary of their address so that they will receive their renewal notices.

January 1—Maine, $4.00. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Sq., Dover-Foxcroft.

January 1—Manitoba, $5.00. Address W. Kurth, D.O., secretary, Board of Osteopathic Physicians, 248 Moorgate Blvd., Winnipeg 12.

January 1—New York, $5.00, biennially. A physician receiving a license the second year of any biennial registration period pays a fee of $2.50 for a certificate expiring December 31 of such second year. Address Stiles D. Ezell, M.D., secretary, Bureau of Professional Ex-

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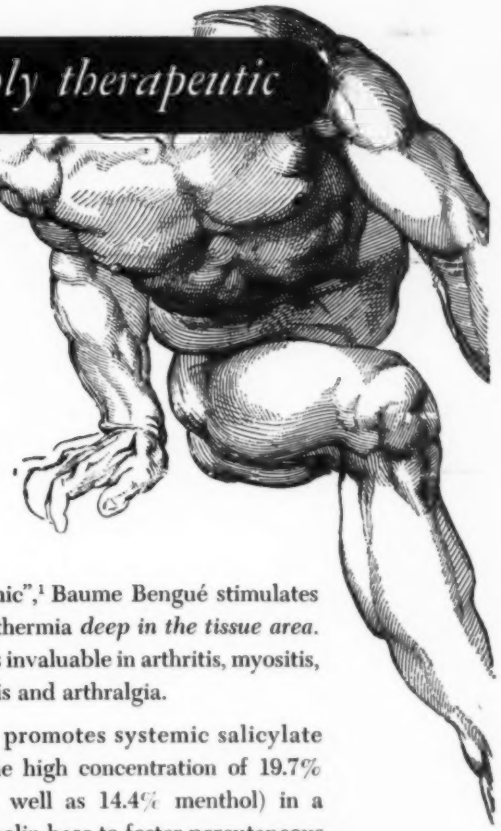

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1. Lange, K., and Weiner, D.: J. Invest. Dermat. 12:263 (May) 1949.

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January 1—Ontario, $35.00. Address D. Gordon Campbell, D.O., secretary, Board of Directors of Osteopathy, 2 Bloor St., E., Toronto 5.

January 1—Pennsylvania, Surgeons Examining Board, $10.00. Address Mrs. Katherine M. Wollet, acting secretary, Board of Osteopathic Examiners, Bureau of Professional Licensing, Harrisburg.

January 1—Texas, $5.00. Address M. H. Crabb, M.D., secretary, Board of Medical Examiners, 1714 Medical Arts Bldg., Fort Worth 2.

January 1—Utah, $3.00. Address Alice E. Houghton, D.O., secretary, Osteopathic Examining Board, 600 Zion's Savings Bank Bldg., Salt Lake City 1.

January—Alberta. No reregistration. Pay $10.00 a year membership in the College of Physicians and Surgeons, Alberta, in January.

During January—Connecticut, $2.00. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

During January—Minnesota, $2.00. Address Wallace F. Kreighbaum, D.O., secretary, Board of Osteopathic Examiners, 2748 Hennepin Ave., Minneapolis 8.

During January—Wisconsin, $3.00. Address Thomas W. Tormey, Jr., M.D., secretary, Board of Medical Examiners, State Office Bldg., 1 W. Wilson St., Madison.

January 31—British Columbia, amount of fee set at Annual Meeting of Council of College of Physicians and Surgeons of British Columbia. Address Lynn Gunn, M.D., Registrar, College of Physicians and Surgeons of British Columbia, 1807 W. 10th Ave., Vancouver 9.

Before February 1—Vermont, $3.00 for residents, $2.00 for nonresidents. Address Charles D. Beale, D.O., secretary, Board of Osteopathic Examination and Registration, Mead Bldg., Rutland.

Before March 1—Colorado, $2.00 if legal resident; $10.00 if not legal resident. Address Samuel H. Brown, M.D., executive secretary, Board of Medical Examiners, 831 Republic Bldg., Denver 2.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene; medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

Part III examinations will be given at the College of Osteopathic Physicians and Surgeons, Los Angeles, March 5-6, at the Chicago College of Osteopathy, Chicago, April 3-4, and in the Spring at Philadelphia and Kirksville, Mo.

These are oral examinations which the candidate may take after having satis-

factorily completed the first 6 months of a 1-year internship in a hospital approved by the American Osteopathic Association for intern training. Part III is given annually at the above-named colleges.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1-year internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.

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UNKNOWNS IN VITAMIN C*

By Leslie J. Harris, D.Sc.†

Among the principal gaps remaining in our knowledge about vitamin C, the three that are especially noticeable relate to requirements, distribution, and mode of action.

VITAMIN C REQUIREMENTS

There is as yet no universal agreement about the amount of vitamin C required daily by man for maintaining health. We do know that as small a quantity of ascorbic acid as 10 mg. per day, or somewhat less, is sufficient to prevent scurvy. But we also know, and it is a familiar observation to everyone who has carried out vitamin assays on animals, that there is generally a wide gap between the minimum and the optimum dose—that is to say, between the relatively small amount of any vitamin needed to prevent actual lesions of frank deficiency disease and the much larger amount needed to promote full health as measured by a maximal growth response in the young animal and by normal physiological function.

Avitaminosis and Hypovitaminosis

With regard to vitamin C, the arguments in favor of the view that the optimum may be widely separated from the minimum can be marshalled under several headings.

Subscurvy in experimental animals. Tests on guinea pigs do indeed prove that the quantity of vitamin C needed to prevent so-called clinical signs of scurvy is considerably less than that necessary to obtain the full potentialities of growth in the young animal or to maintain, for example, the normal functioning of the odontoblasts. The curve of response is in fact logarithmic.

Presumptive advantages of saturation. Again, it has been argued that, so far as is known, all animal species except primates and guinea pigs can synthesize vitamin C in the body and do not need it in their diet, and that it seems a noteworthy fact that in such species Nature has arranged to maintain a state of saturation. Therefore, it is not unreasonable to suppose, as King and others have, that for human beings likewise a state of saturation may be both natural and desirable. There is reason to think, for example, that an intake of vitamin C appreciably above the mere minimum (or scurvy-preventing dose) is needed to promote the optimal healing of wounds.

Absence of direct proof. On the other side, we are forced to admit that how-

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†Dr. Harris is director of the Dunn Nutritional Laboratory, University of Cambridge and Medical Research Council, and president of the Nutrition Society. This paper is based on his summary remarks at the Lind Bicentenary Symposium conducted at the University of Edinburgh last May in honor of Dr. James Lind, author of the Treatise of the Scurvy published in Edinburgh in 1753. The Proceedings of the Nutrition Society, published by the Cambridge University Press, London, carries the full text of the symposium.

ever attractive the presumptive evidence may seem, or however useful a safeguard it undoubtedly is to provide a liberal intake of vitamin C, if only to insure a reasonable margin of safety, nevertheless, we have as yet no means of definitely proving that a man is in better health for receiving 30 mg. of ascorbic acid daily, the League of Nations standard of requirement *(1)*, instead of 10 mg. per day, the approximate scurvy-preventing dose. We have still less direct evidence for insisting on an allowance of up to 50 or 75 mg. to meet some of the proposed American standards *(2)*.

Need for functional tests. The one thing that would help more than anything else in settling this controversy about the requirements would be to have new functional tests developed to demonstrate the effect of minor degrees of deficiency. Whereas for some other vitamins such tests are already in existence, for vitamin C they are still lacking and are most urgently needed. An example of the kind of test I have in mind is the measurement of dark adaptation for vitamin A, which permits the diagnosis of a mild degree of deficiency of this vitamin before any clinical signs have become evident. Similarly, for vitamin B_1, the carbohydrate tolerance (pyruvate clearance) test originated by Banerji and myself *(3)* and applied by McCollum and others in America and elsewhere, will give evidence of some impairment in vitamin B_1 function when the deficiency is still too slight to produce the more obvious clinical manifestations of disease *(4-7)*.

Likewise, for vitamin C we must look forward to seeing some functional stress-test devised. Possibly, bearing in mind Dr. Crandon's observations *(8)*, it may be some kind of a local stress reaction. Only when such tests are available will it become possible to detect minor degrees of deficiency in man and hence to know what is his real optimal requirement.

DISTRIBUTION

Another outstanding puzzle about vitamin C is to know why it is present in such relatively large amounts in some fruits, or in certain plant and animal tissues, and yet is almost entirely absent from other, and apparently analogous, fruits or tissues. This unevenness in its distribution would seem to be almost fortuitous. Are we then to inquire teleologically what is the purpose of these large amounts of vitamin C in some sites and the smaller amounts in others closely similar; or are we to regard it, as it almost appears to be, as just a biochemical accident? We can do little more than state the question, for there appears as yet to be little or no hint of the answer.

MODE OF ACTION

We are still largely in the dark also about the physiology and biochemistry of vitamin C action. We cannot yet point to any precisely defined coenzyme system, such as is possible with various vitamins of the B group.

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The Known Facts

What is known about vitamin C action can be summarized (for the greater part) under three headings, as follows:

Reducing action. Although it has been shown that vitamin C, by reason of its reducing activity, is able to participate in, or to stimulate, a large number of chemical reactions, this action seems largely unspecific, for other reducing agents can often replace the vitamin. In this direction, then, we are not much nearer explaining the characteristic action of the vitamin in preventing scurvy.

Structural effects of deficiency. All that need be said under this heading is that in the absence of adequate amounts of vitamin C the formative cells of the body, such as the odontoblasts, ameloblasts, cementoblasts, and osteoblasts, lose their normal functional activity and cease to lay down the normal type of new tissue, namely, dentine, enamel, cement, and bone. A particular instance of this is the well-known fact that with a deficiency of vitamin C the normal production of collagen is impaired.

Specific chemical effects. It is clearly

1. Beckman, H. *Treatment in General Practice* (Saunders) 1948. 2. Krantz, J. C. & Carr, C. J.: *The Pharmacologic Principles of Medical Practice* (Williams & Wilkins) 1951.

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established that vitamin C is involved in two distinct chemical reactions, or systems, namely: (*a*) the conversion of folic acid to folinic acid, and (*b*) the metabolism of tyrosine and related substance. An additional point here, as suggested by the new findings of King (*9*), is that in vitamin C deficiency cholesterol metabolism may also be affected. Apart from this, little is definite.

Adrenal Function

The fact that the administration of a dose of ACTH is followed by a temporary fall in the level of ascorbic acid in the adrenal gland (*10*) suggests that vitamin C may be related in some way to adrenal function. This idea is further supported by the consideration that vitamin C is present in extraordinarily high concentration in the adrenal, both in the medulla and the cortex. However, the attempts of my colleagues and myself to find evidence of a consistent relation between ACTH (or cortisone) function and vitamin C requirements have so far led to essentially negative conclusions (*11*). According to C. P. Stewart and others (*12,13*), ACTH and cortisone may affect the equilibrium between ascorbic and dehydroascorbic acids in the blood of human subjects. Yet our own tests failed to show any essential change in the ratio of ascorbic acid to dehydroascorbic acid in the adrenals of guinea pigs after an injection of ACTH.

CONCLUSION

From what has been said, it would seem helpful, then, if in our discussions and in future work, some particular consideration could be given to these three problems: (1) the quantitative requirements for vitamin C, (2) the reason for its apparently uneven distribution, and (3) the detailed nature of the specific biochemical system or systems in which the vitamin functions and the derangement of which presumably leads to scurvy.

REFERENCES

(*1*) League of Nations: Reports by the Technical Commission on Nutrition on the work of its third session held in London, November 15-20, 1937. Bull. Health Org., League of Nations 7:460-502 (1938).

(*2*) National Research Council. Food and Nutrition Board: Recommended dietary allowances. Reprint and Circular Series No. 129. Washington, D. C., The Council, 1948. Revised. Also Nutrition Rev. 6:319 (1948).

(*3*) Banerji, G. G., and Harris, L. J.: Methods of assessing level of nutrition. Carbohydrate tolerance test for vitamin B_1; experiments with rats. Biochem. J., Lond. 33:1346-1355 (1939).

(*4*) Shils, M. E., Day, H. G., and McCollum, E. V.: Effect of thiamine deficiency in rats on excretion of pyruvic acid and bisulfite-binding substances in urine. J. Biol. Chem. 139:145-161 (1941).

(*5*) Bueding, E., Stein, M. H., and Wortis, H.: Blood pyruvate curves following glucose ingestion in normal and thiamine-deficient subjects. J. Biol. Chem. 140: 697-703 (1941).

(*6*) Harris, L. J.: Biochemistry. 2. Nutrition and vitamins. *In* Annual Reports on the Progress of Chemistry for 1940. Vol. 37. London, The Chemical Society, 1941.

(*7*) Williams, R. D., Mason, H. L., and Wilder, R. M.: Minimum daily requirement of thiamine of man. J. Nutrition 25:71-97 (1943).

(*8*) Crandon, J. H., Mikal, S., and Landeau, B. R.: Ascorbic-acid deficiency in experimental and surgical subjects. Proc. Nutrition Soc. [London] 12:273-279 (1953).

(*9*) King, C. G.: The discovery and chemistry of vitamin C. Proc. Nutrition Soc. [London] 12:219-227 (1953).

(*10*) Sayers, G., Sayers, M.A., Lewis, H. L., and Long, C. N. H.: Effect of adrenotropic hormone on ascorbic acid and cholesterol content of adrenal. Proc. Soc. Exper. Biol. & Med. 55:238-239 (1944).

(*11*) Harris, L. J., Bland, M. N., Hughes, R. E., and Constable, B. J.: Effect of A. C. T. H. and cortisone on vitamin C metabolism and on the weight and composition of the liver in guinea pigs and other species. Lancet [London] 1:1021-1024 (1953).

(*12*) Stewart, C. P., Horn, D. B., and Robson, J. S.: Dehydroascorbic acid in human blood plasma. Proc. Nutrition Soc. [London] 12:300-305 (1953).

(*13*) Stewart, C. P., Horn, D. B., and Robson, J. S.: The effect of cortisone and adrenocorticotropic hormone on the dehydroascorbic acid of human plasma. Biochem. J. [London] 53:254-261 (1953).

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1. Cass, L. J. and Frederik, W. S.: Malt Soup Extract as a Bowel Content Modifier in Geriatric Constipation. Journal-Lancet, 73:414 (Oct.) 1953.

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PROGNOSIS IN RHEUMATIC FEVER*

The mortality from rheumatic fever, which has been decreasing for many years, declined even more rapidly in the past decade. This recent improvement has been due in part to general betterment in environmental and living conditions, and more particularly to the greater control over streptococcal infections with sulfa drugs, penicillin, and other antibiotics. The new therapy has been useful in preventing both initial and recurrent attacks of rheumatic fever.

It is still too early to evaluate the long-term outlook for children who have suffered a first attack of rheumatic fever in recent years. Fortunately, the number of such children definitely appears to be declining. Valuable information on the outlook for patients coming under treatment 15 or more years ago is available from several recent studies based upon the experience of insured lives.

One of these is a follow-up study of the mortality among a group of nearly 3,000 children with rheumatic fever insured under Industrial policies in the Metropolitan Life Insurance Company. These policyholders, who were in the age range from 1 to 20 years when they first received nursing care during an acute attack some time in the period 1936 to 1938, have been systematically traced since that time. The majority of these children were still living at the end of 15 years. The maximum survivorship rate—94 percent—was recorded among girls under age 10 at the time of attack who had no evident heart disease reported at first observation. Even the minimum survivorship rate over the 15-year period, that among boys with heart disease reported during the attack, was close to 65 percent.

As might be expected, the mortality was highest in the early years after the attack, especially for those with heart involvement. For example, among boys with heart involvement the death rate in the first five years after the attack averaged 66 per 1,000, compared with 17 in the next five years and with 8 per 1,000 in the succeeding five years. In part at least, the recent improvement reflects the prevention of recurrent attacks of the disease.

The mortality among the children under study during the 15-year period was nevertheless well above normal, primarily because of the resulting heart damage. Rheumatic heart disease accounted for more than three fifths of all deaths. Among children with heart involvement in the initial attack, the proportion of deaths due to rheumatic heart disease exceeded three fourths, and was especially high among the younger children. Even among children without reported heart involvement during the acute stage, the majority of deaths occurring subsequently were due to rheumatic heart disease.

Of particular interest clinically is the record for subacute bacterial endocarditis. Altogether, 10 percent of the deaths were ascribed to this cause. The mortality from subacute bacterial endocarditis was largely concentrated in the early years of the experience. At the time this study was inaugurated, it was expected that mortality from this condition, which then invariably ran a fatal course, would increase as time went on. That this did not occur reflects, no doubt, the influence of the antibiotics both in prevention of recurrent attacks of rheumatic fever and in the treatment of subacute bacterial endocarditis. This is one of the most significant changes in the prognosis of rheumatic fever.

Even though the majority of the girls in this study have reached marriageable age, and a large proportion are married, there have been only two deaths from complications of pregnancy and childbirth among them. One of these deaths followed the birth of twins, an event which normally involves extra risk to the mother.

The various studies of rheumatic fever indicate that the prognosis of the disease has improved, with prospects of complete recovery in a high proportion of cases. Early diagnosis and proper treatment are

*Reprinted from *Statistical Bulletin*, Metropolitan Life Insurance Company, August, 1954.

still of prime importance for a favorable outcome. Even persons with severe heart damage resulting from rheumatic fever are now being treated successfully. Surgical correction of such defects is becoming increasingly frequent, and a growing number of patients with rheumatic heart disease are being restored to health and working capacity. For some time now cortisone and ACTH have been under careful trial in the treatment of rheumatic fever. It is hoped that this hormonal treatment will prove effective in lessening or preventing permanent heart damage from rheumatic fever, but this therapy awaits definitive evaluation.

MORBIDITY AND MORTALITY TRENDS IN POLIOMYELITIS*

In each of the past six years the reported incidence of acute poliomyelitis in the United States was higher than at any time in the past. The number of cases reached an all-time peak of nearly 58,000 in 1952, and last year, with about 36,000 cases, was the third highest on record; these figures compare with somewhat over 27,000 cases reported in the major epidemic year 1916. During the quarter century from 1917 to 1942, in only three years did the reported cases of poliomyelitis exceed 10,000; since then, every year exceeded that figure without exception.

In contrast to the rise in morbidity are the reassuring facts on the trend of mortality. In the past decade the average death rate was below that in the first two decades of the period under review. Even the peak death rates in recent years were less than one tenth the rate of 36.8 per 100,000 recorded in 1916.

Several difficulties are encountered when one attempts to interpret the morbidity and mortality trends in poliomyelitis for the country as a whole. The incidence of the disease fluctuates widely from year to year, as does its geographic distribution. Adequate reporting of cases on a nationwide scale began little more than 20 years ago, and reporting practices have by no means been uniform. Until fairly recent years relatively few States systematically reported cases by age or severity.

Some of the factors known to influence the trend in poliomyelitis throw light on the situation. For example, the rise in the number of cases reflects the growth of the child population, but this accounts for only a relatively small part of the increase in the incidence of the disease. As against an increase of about 400 percent in cases between 1930-1934 and 1949-1953, the population under age 10 has increased less than 35 percent and at ages 10-19 there was actually a small decrease. Over the past 20 years an increasing proportion of the cases and the deaths have been occurring at the later childhood ages and in early adult life; yet even now, more than two thirds of all reported cases are at ages under 15.

The contrasting trends of morbidity and mortality reflect, to some extent, the increased proportion of nonparalytic cases, but this too is apparently not a major factor. The areas for which the necessary data are available show only a moderate increase in nonparalytic cases. Moreover, the proportion of nonparalytic cases reported has always tended to be higher in epidemic than in non-epidemic years.

One of the significant characteristics of the disease has been its shifting geographic pattern. The recent rise in the frequency of poliomyelitis has been relatively greatest in States west of the Mississippi River, smallest in the Northeastern part of the country, while the Middle West and South occupy an intermediate position in this respect. There are, however, wide variations in the trend of poliomyelitis not only within broad geographic regions but also within individual States.

The reporting of cases was incomplete in earlier years, but in recent years there has been some over-reporting of the disease. The wide publicity that has been given poliomyelitis, coupled with the un-

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Copper	0.1 mg.	*
Cobalt	0.1 mg.	**
Nickel	0.1 mg.	**
Magnesium	1 mg.	*
Potassium	5 mg.	*
Molybdenum	0.1 mg.	**
Zinc	0.1 mg.	**
Fluorine	0.1 mg.	**
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Phosphorus	120 mg.	16%
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(1) Identified by some as whole natural Vitamin P Complex. (2) See ingredients. (*) Amount required in human nutrition not established. (**) Need in human nutrition not established.

derstandable fear of the disease, has made both parents and physicians very conscious of it. Consequently, children with presumptive symptoms are sometimes reported as having poliomyelitis and are counted in the case records although the subsequent facts do not justify the initial diagnosis. Unfortunately, such case reports are not subsequently corrected, nor are there ways and means to attempt this on a country-wide basis. In New York City, review by the Health Department of the original diagnosis indicated that the over-reporting of cases has amounted to about 15 percent during the past few years.

Among the important factors accounting for the increase in the incidence of the disease in our country appears to be a change in the age at which natural immunity to the disease is acquired. It seems likely that as a result of factors that reduce close personal contact among children—such as the mobility of the population, the decrease in the size of families, and the growth of suburban areas—the acquisition of natural immunity has been reduced or delayed. However, the significance of this is rather small, for even under current conditions a large majority of children build up immunity at a fairly early age. Consequently, even in recent years the highest annual incidence of reported cases at ages under 15 has been only one per 1,000. The chances that a person would be struck by the paralytic type of the disease before he reaches 20 are less than one in 500. Likewise, the annual death rates from the disease in recent years have averaged about 2.5 per 100,000 at the childhood ages, while the chances of dying from the disease between birth and age 20 are only about one in 2,000.

Intensive investigations are being directed to the prevention of poliomyelitis, with attention at the moment being focused on the new vaccine now under large-scale trial. At the same time, more and more is being accomplished in the treatment and rehabilitation of those stricken as knowledge of the disease advances and as trained personnel and facilities for care of patients increase.

Books Received

UROLOGY. Edited by Meredith Campbell, M.S., M.D., F.A.C.S., Emeritus Professor of Urology, New York University. Cloth. 3 Vols. Pp. 2356, with illustrations. Price $60.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

THE CLINICAL USE OF CORTICOTROPIN, CORTISONE AND HYDROCORTISONE IN EYE DISEASE. By Dan M. Gordon, M.D., F.A.C.S., Assistant Professor, Department of Surgery (Ophthalmology), New York Hospital-Cornell Medical Center, New York. Cloth. Pp. 88, with illustrations. Price $3.75. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

FLUID AND ELECTROLYTES IN PRACTICE. By Harry Statland, M.D., Associate in Medicine, University of Kansas School of Medicine; Consultant in Medicine, Veterans Administration Hospital, Kansas City, Missouri; Attending Physician, Menorah Medical Center, Kansas City, Missouri. Cloth. Pp. 206, with illustrations. Price $5.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

The Clinical Significance of DISTURBANCES IN THE DELIVERY OF SWEAT. By Marion B. Sulzberger, M.D., F.A.C.P., and Franz Herrmann, M.D., Department of Dermatology and Syphilology, New York University Post-Graduate Medical School; Skin and Cancer Unit of University Hospital, New York University-Bellevue Medical Center. Paper. Pp. 212, with illustrations. Price $6.75. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

STELLATE GANGLION BLOCK, Techniques—Indications—Uses. By Daniel C. Moore, M.D., Director, Department of Anesthesiology, Mason Clinic; Chief of Anesthesia, Virginia Mason Hospital, Seattle, Washington. Cloth. Pp. 280, with illustrations. Price $10.50. Charles C Thomas, Publisher, Springfield, Ill., 1954.

PRINCIPLES OF INTERNAL MEDICINE. Edited by T. R. Harrison, Raymond D. Adams, William H. Resnik, Paul B. Beeson, George W. Thorn, and M. M. Wintrobe. Ed. 2. Cloth. Pp. 1703, with illustrations. Student price, 1 Vol. Ed., $16.00. Professional price, 2 Vol. Ed., $21.00. The Blakiston Company, 575 Madison Ave., New York 22, 1954.

A HISTORY OF MEDICINE. By Ralph H. Major, M.D., Professor of Medicine and of the History of Medicine, The University of Kansas, The School of Medicine, Kansas City, Kansas. 2 Vols. Cloth. Pp. 1056, with illustrations. Price $14.50. Charles C Thomas, Publisher, Springfield, Ill., 1954.

COLOR ATLAS OF PATHOLOGY. Endocrine System, Including Pituitary, Thyroid, Parathyroid, Adrenals and Pancreas; Gynecology and Obstetrics, Including Reproductive Organs; Breasts; Male Genital Tract; Skin. Prepared under the auspices of the U.S. Naval

Medical School of the National Naval Medical Center, Bethesda, Maryland. Cloth. Pp. 450, with illustrations. Price $20.00. J. B. Lippincott Company, East Washington Square, Philadelphia 5, 1954.

MODERN DIAGNOSIS AND TREATMENT OF THE MINOR VENEREAL DISEASES. By Orlando Canizares, M.D., F.A.C.P., Associate Professor, Clinical Dermatology and Syphilology, New York University, Post-Graduate Medical School; Consultant, Veterans Administration Hospital, Bronx, New York; United States Public Health Service Hospital, Staten Island, New York. Cloth. Pp. 121, with illustrations. Price $3.75. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

PROGRESS IN CLINICAL SURGERY. Edited by Rodney Smith, M.S., F.R.C.S., Surgeon, St. George's Hospital, London. Cloth. Pp. 414, with illustrations. Price $5.00. Little, Brown & Company, Boston 6, 1954.

THE PYRAMIDAL TRACT, Its Status in Medicine. By A. M. Lassek, M.D., Ph.D., Professor of Anatomy, Boston University School of Medicine, Boston, Massachusetts. Paper. Pp. 166. Price $4.75. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

ELEMENTS OF PEDIATRIC ANESTHESIA. By C. R. Stephen, B.Sc., M.D.C.M., F.A.C.A., D.A (R.C.P. &S.) London, England, Professor of Anesthesiology, Duke University; Chief, Division of Anesthesia, Duke University Hospital, Durham, North Carolina; Formerly, Chief, Department of Anesthesia, Children's Memorial Hospital, Montreal, Quebec, Canada. Paper. Pp. 109, with illustrations. Price $3.50. Charles C Thomas, 309-327 E. Lawrence Ave., Springfield, Ill., 1954.

DISEASES OF THE SKIN For Practitioners and Students. By George Clinton Andrews, M.D., F.A.C.P., Clinical Professor of Dermatology, College of Physicians and Surgeons, Columbia University; Attending Dermatologist to the Presbyterian Hospital, Columbia-Presbyterian Medical Center, New York. Ed. 4. Cloth. Pp. 877, with illustrations. Price $13.00. W. B. Saunders Company, West Washington Square, Philadelphia 5, 1954.

NERVE BLOCKS. A Manual of Regional Anesthesia for Practitioners of Medicine. By John Adriani, M.D., Director, Department of Anesthesiology, Charity Hospital of Louisiana; Professor of Surgery, Tulane University School of Medicine; Associate Clinical Professor of Surgery, Louisiana State University School of Medicine, New Orleans, Louisiana. Cloth. Pp. 265, with illustrations. Price $6.50. Charles C Thomas, Publisher, Springfield, Ill., 1954.

PRINCIPLES OF OSTEOPATHIC TECHNIC. By Harrison H. Fryette, D.O. A Harry L. Chiles Memorial Publication. Pp. 241, with illustrations. Academy of Applied Osteopathy, Carmel, Calif., 1954.

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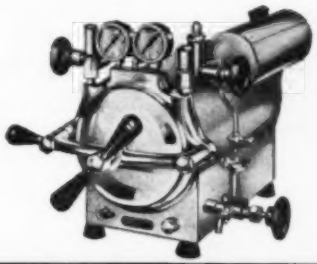

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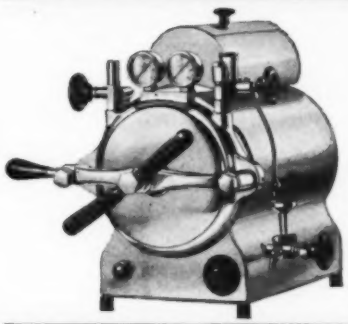

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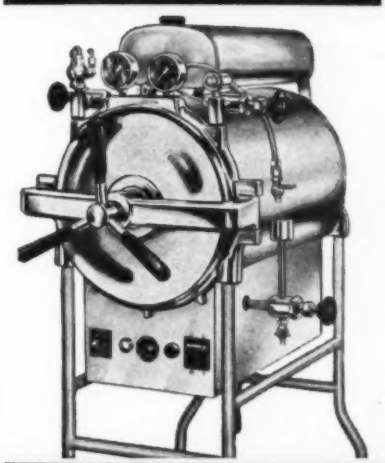

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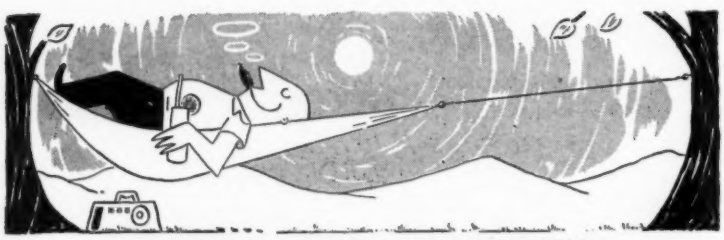

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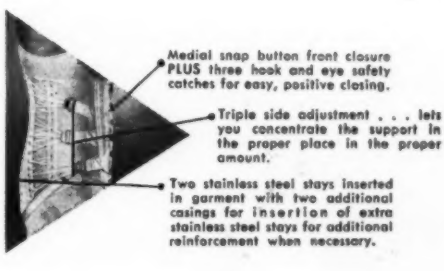
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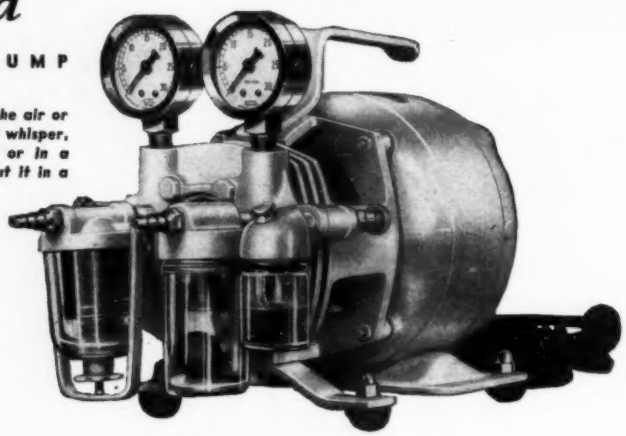

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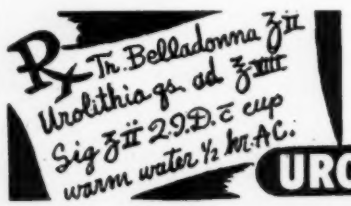

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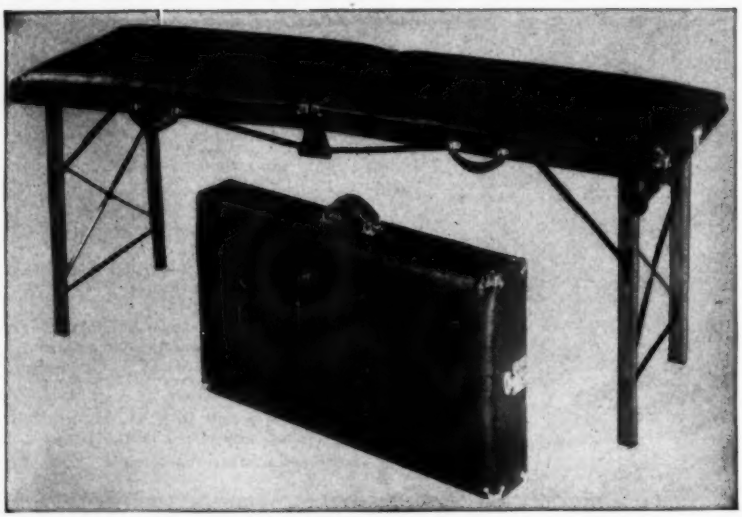

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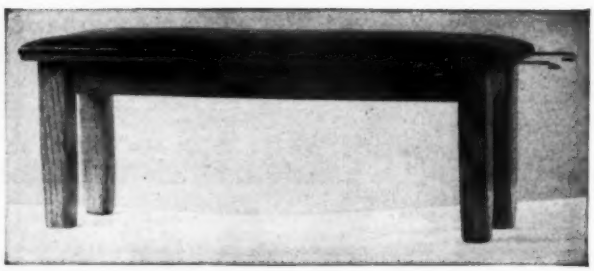

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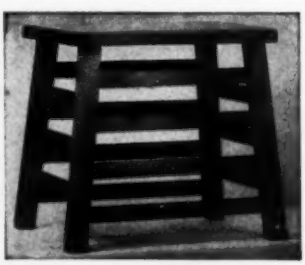

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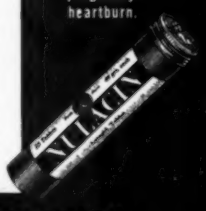

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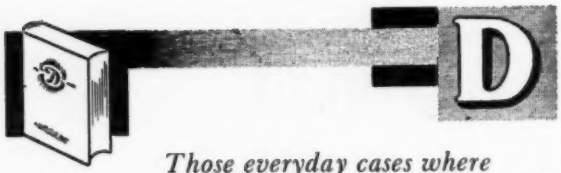

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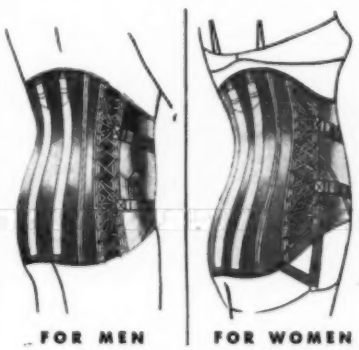

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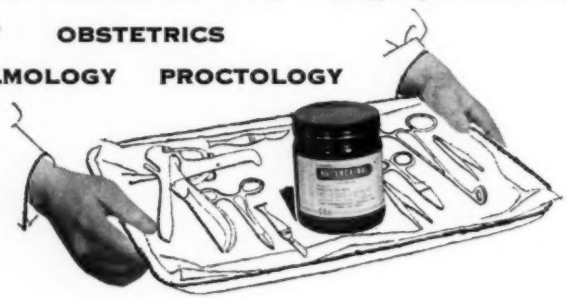

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